

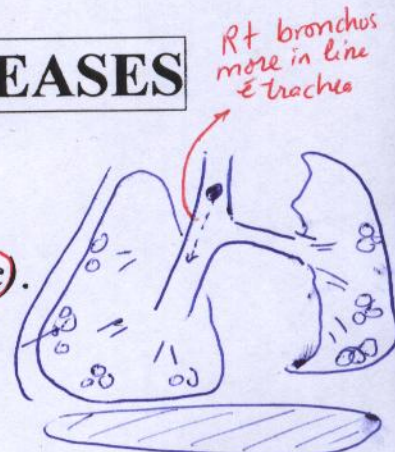
INTRODUCTION

CLASSIFICATION OF CHEST DISEASES

1. BRONCHIAL DISEASES: (4)

- Bronchitis (B.A. itis)
- Bronchial asthma (B.A. itis)
- Bronchiectasis (B.A. itis)
- Bronchogenic carcinoma (Ca.)

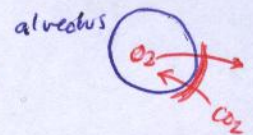
(acute) & (chronic).



2. PARENCHYMATOUS LUNG DISEASES: (5)

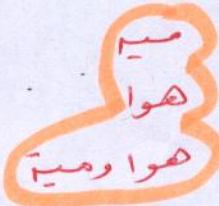
- Consolidation (pneumonia).
- Cavitation (lung abscess).
- Collapse.
- Fibrosis.
- Emphysema.

alveoli + B.V. + interstitial tissue



3. PLEURAL DISEASES: (4)

- Pleurisy. (itis)
- Pleural effusion.
- Pneumothorax.
- Hydropneumothorax.



4. MEDIASTINAL DISEASES.

5. DIAPHRAGMATIC DISEASES.

6. CHEST WALL DISEASES.

Most common cause is Bronchogenic Ca. infiltrates phrenic N.

VISIT...

LANZAROTE
Caliente.COM

CHEST SYMPTOMS

COUGH

- It is the **most common** symptom of chest diseases.
- It is a **defensive** mechanism aiming at expulsion of secretions or inhaled particles from the respiratory tract.

Hemoptysis
Acute dyspnea
Chest Pain
Hazards of cough

التهاب
2- P-
التي نظري
منه ياتو

ETIOLOGY

I. Reflex cough:

Respiratory

others

(due to irritation of cough receptors)

A. Respiratory causes:

1. Pharyngeal diseases:

- 4 - Causes: pharyngitis, post-nasal discharge, tonsillitis, tumours.
- Features: painful, & may be accompanied by nausea & vomiting.

2. Laryngeal diseases:

- 3 - Causes: laryngitis, foreign body, tumours.
- Features: painful, & may be accompanied by stridor & hoarseness of voice.

3. Tracheal diseases:

- 1 - Causes: tracheitis.
- Features: painful, dry.

4. Bronchial diseases:

- 4 - Causes: bronchitis, bronchial asthma, bronchial tumours, bronchiectasis.
- Features: dry or productive + specific features to each disease.

5. Parenchymatous lung diseases:

- 5 - Causes: pneumonia, lung abscess, collapse, TB, IPF, emphysema.
- Features: dry or productive + specific features to each disease.

6. Pleural diseases:

- 4 - Causes: pleurisy, pleural effusion, pneumothorax, hydropneumothorax.
- Features: dry cough or productive + specific features to each disease. [Pain]

Oral

as irritation of pleura not lung

except one may be with sputum

if
Broncho
Pleural
Fistula
airway de facto

Empyema (pus)

irritates pleura
dry

B. Extra-respiratory causes:

1. Cardiovascular diseases:

- Pulmonary congestion, left sided heart failure (dry or productive cough).
- Pulmonary embolism.

2. Mediastinal causes:

- Causes of mediastinal syndrome

3. Other causes:

- Abdominal causes:

e.g. subphrenic abscess.

irritates basal pleura

eg amoebic

= loud & harsh (brassy cough). due to irritation of trachea & main bronchi

by mediastinal mass



(due to stimulation of the cough center)



II. Central cough:

- Brain tumours,

encephalitis, inflamm. of brain

CVS. stroke

in medulla

III. Psychogenic (Hysterical) cough:

- Usually: in young neurotic females.
- Usually: dry cough, occurring in front of the audience.

HAZARDS OF COUGH

I. Thoracic:

1. Rupture of a pleural bleb, causing pneumothorax.
2. Rupture of bronchial varices.
3. Rupture of an aneurysm.
4. Stress fracture of a rib.

COPD B.A. [in complications of cough]

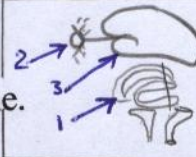
لو حزنه
عنه تفرغ
hemoptysis

if metastasis
or if osteoporotic

II. Extrathoracic:

1. Increased intra-abdominal pressure:

- Hernia
- Rectum
- Bladder
- Hernia.
- Rectal prolapse.
- Stress incontinence of urine.



2. Increased intra-ocular pressure:

- hge detach
- Retinal & subconjunctival hemorrhages.
- Retinal detachment.

3. Increased intra-cranial pressure:

- hge
- SAH.

if congenital aneurysm
or if mycotic aneurysm (I.E)
if atherosclerotic aneurysm

لذلك لازم
جه علينا
لديكون هناك
كحة

bleb = localized collection of air closed sac of air

ساعات واحد يتولد وعنده

هذا ال bleb

في أي حتمه lung

لكنه غالباً فوقه apical

لو تفرغ pneumothorax



HEMOPTYSIS

DEFINITION

- * Coughing of blood originating from: below the vocal cords.
- Bleeding originating from above the vocal cords is known as: false hemoptysis.
- Hemoptysis may be: frank or blood tinged.

ETIOLOGY

Respiratory & CVSE & systemic

(1) Respiratory causes:

A. Laryngeal causes:

- Inflammation.
 - Tumours.
- damage of mucosa & caps
eg acute bronchitis
1) lge & necrosis
2) neovascularization

B. Tracheo-bronchial causes:

- Acute Bronchitis. (the most common cause)
 - Bronchiectasis. (inflammation + dilatation)
 - Bronchial carcinoma.
 - Bronchial adenoma.
- inflammation destroys caps
Force of cough ruptures caps

C. Pulmonary causes:

- Pulmonary TB. (not most common)
 - Pulmonary embolism.
 - Pneumonia.
 - Lung abscess.
 - Goodpasture syndrome.
- Bleeding of vascular granular tissue
erosion of B.v. crossing cavity
inflammation → injures caps of mucosa
autoimmune disease
Abs against lung: alveolitis: Interstitial pulmonary fibrosis
Kidney: glomerular b.m. (G.N.)
Vasculitis: hemoptysis
lung disease
long standing - it is in alveoli
fibrosis

(2) Cardiovascular causes:

- Pulmonary congestion, e.g. MS & acute pulmonary oedema.
- Pulmonary infection.
- Pulmonary infraction.
- Rupture of bronchial varices.
- Rupture of aortic aneurysm.

(3) Systemic diseases:

Hemorrhagic blood diseases, e.g.

- Clotting factors • Hemophilia.
- Platelets • Purpura.

الباتة يتوجع
Blood

ويتركز فيه
Bleeding from
other
orifices

generalized
not
localized

لذلك لازم تسأل الطبيب فيه نزيف
White Knight Love
نصفه ولا
هتلاينه

3

MANAGEMENT OF HEMOPTYSIS

I. Diagnosis:

- Past H/O
- Present H/O
- Blood (attack)
- After attack
- C/P
- Investig

(1) Differentiation between hemoptysis and hematemesis:

	Hemoptysis	Hematemesis
Past history	Chest disease	GIT disease
The attack	Cough	Vomiting
Blood Attack	Bright red, frothy	Dark red, food particles
After the attack	Streaked sputum	Melena tarry
Examination	Chest signs	Abdominal signs
Investigations	Chest or heart disease	GIT disease

exclude

Other Types

(2) Exclusion of false hemoptysis:

- Examination of the upper respiratory tract usually reveals the cause.

(3) Detection of the cause of hemoptysis:

A) Clinical picture:

- Symptoms & Signs.

B) Investigations:

1. Chest X-ray.
2. CT chest.
3. Cardiac investigations: ECG & Echocardiography.
4. Bronchoscopy & biopsy for (eg: Bronchogenic Ca.)
5. Blood picture.
6. Bleeding profile.
7. SPUTUM EXAMINATION.

Tests

1. CXR
2. CT
3. Broncho sputum
4. Bld Pic
5. Bld Profile
6. Cardiac
7. Cardiac

Oral exception
GIT bright red
bright red

yes: ① if massive

② if no HCl

& achlorhydria

③ if on antacids for long time

B.T
CT
PT, T.T
APTT

Bleeding Time / Clotting Time / Prothrombin Time / Thrombin Time / APTT

II. Treatment:

1. Treatment of the cause.

2. Cases of massive hemoptysis require:

- Hospitalization.
- Sedation.
- Blood transfusion & anti-shock measures.

How can sputum analysis help?

- TB: T.B. bacilli
- Br. Ca: malignant cells
- pneumonia: organism
- B.A.

if hemodyn. stable

if hemodyn. unstable

المزيج
من
الدم
و
الغذاء
من
الدهن
و
السكر
و
البروتين

CHEST PAIN

نظرة واحدة
symp واحد

ETIOLOGY

I. Cardiovascular causes:

- ♣ **Myocardium:**
Coronary heart disease: angina or infarction.
- ♣ **Pericardium:**
Pericarditis or pericardial effusion. not acute
- ♣ **Endocardium:**
Mitral valve prolapse.
- ♣ **Aorta**
Aortic aneurysm, & aortic dissection.
- ♣ **Pulmonary:**
Pulmonary embolism & infarction.
- ♣ **Huge cardiomegaly.**
- ♣ **Pain of cardiac neurosis.**

II. Chest causes:

- 1 o Pleural disease: pleurisy, pleural effusion, pneumothorax, Hydropneumothorax
- 2 o Pulmonary disease extending to the pleura, e.g., pneumonia & lung abscess.
- 3 o Acute massive lung collapse.

III. Mediastinal causes:

- o Oesophagus: oesophageal spasm, oesophagitis, GORD = acid in wrong place
 - o Mediastinal tumours.
 - o Mediastinal lymphadenopathy
 - o Mediastinitis.
- Commonest cause iatrogenic: perforated jejun. in oesophagoscopy
" " Bronchus in Bronchoscopy*

IV. Chest wall causes:

- o Skin: wounds.
 - o Breast: mastitis, tumour.
 - o Ribs: osteomyelitis, tumour, fracture.
 - o Intercostal muscles: myositis, muscle strain (from severe cough).
 - o Intercostal nerves: nerve root pain (herpes zoster).
- Tietze's syndrome (oral)
Costo-chondritis
unknown cause
viral غالباً*
- Burning 3-4d → skin rash vesicular
① in already immunocompromised
② neurotropic virus
③ Cr. Nerve V
I.C. N.N. → يحس كآبة في
ولا يتنظ إلا لو imm*

V. Abdominal causes:

- o Gastritis, peptic ulcer, hiatus hernia.
- o Gall bladder disease.

CAUSES OF ACUTE CHEST PAIN

وکتب
Criteria each

- (1) MYOCARDIUM: angina pectoris & acute myocardial infarction.
- (2) PERICARDIUM: acute pericarditis.
- (3) ENDOCARDIUM: mitral valve prolapse.
- (4) AORTA: dissecting aneurysm of the aorta.
- (5) PULMONARY: massive pulmonary embolism & pulmonary infarction.
- (6) CARDIAC NEUROSIS.
- (7) Pleural disease: acute pleurisy. ← Pneumothorax
- (8) Lung disease: acute massive lung collapse & pneumothorax.
- (9) Oesophageal disease: oesophageal spasm, oesophagitis, GORD.
- (10) Nerve root pain: herpetic neuralgia.
- (11) Musculo-skeletal disorders: e.g. Tietze syndrome.

DYSPNEA

ETIOLOGY

1. Cardiovascular diseases

- Pulmonary congestion: *بعض القلوب*
- Left-sided heart failure: MS & Left ventricular failure.
- Pulmonary embolism.
- Pericardial effusion. (*Prayer's*)

2. Chest diseases

- Laryngeal causes:
- Foreign body, tumours.] **Obstruction**
- Tracheo-bronchial causes:
- Chronic obstructive lung disease.
- Bronchial asthma.
- Bronchiectasis.
- Bronchial tumours.] **Obstructive lung disease**
- Lung causes:
- Consolidation. *Pneumonia*
- Collapse.
- Fibrosis.
- Interstitial lung disease (ILD).
- Consolidation = part of lung in w air is replaced by inflamm. fluid & infl. cells
- both ↓ elasticity
- ↓ lung compliance
- Pleural causes:
- Pleurisy, pleural effusion, pneumothorax, hydropneumothorax.] **Restrictive**
تقييد حركة الرئة
- Chest wall:
- Obesity.
- Kyphoscoliosis.
- Pain → limits movement
- underlying lung disease
- Limitation of chest wall movement (Compliance ↓)

*3. General diseases

- Anemia.
 - Haemorrhage & shock.
 - Acidosis.
- Respiratory Hs (weak)
+++ R.C. (by periph. chemoreceptors)
tissue hypoxia ↓ O₂
++ RC by both peripheral chemoreceptors or central " "

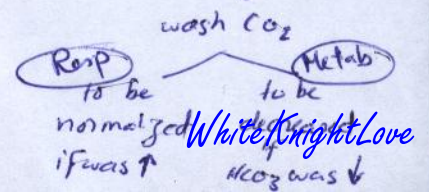
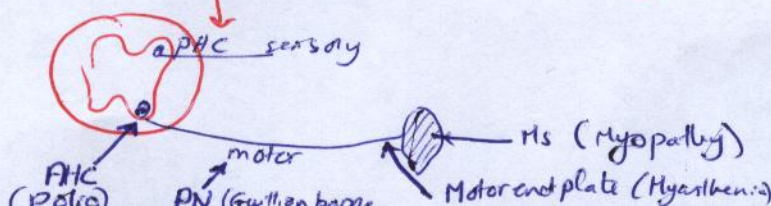
4. Abdominal diseases

- Abdominal distension, e.g. marked ascites.

*4. Neurological diseases

- Vocal cord paralysis.
 - Diaphragmatic paralysis. → *bronchogenic carcinoma injure phrenic N.*
 - Peripheral neuropathy, myasthenia gravis & myopathy.
 - Poliomyelitis affecting respiratory muscles.
- restrictive*

5. Psychogenic (Hysterical) dyspnea. *young neurotic ♀*



CAUSES OF ACUTE DYSPNEA

دyspnea

1. **Acute** left-sided heart failure, e.g. Acute myocardial infarction.
2. **Acute** pulmonary embolism.
3. **Acute** massive lung collapse.
4. Tension **P**neumothorax.
5. **P**neumonia. oral may be chronic rarely
6. **P**leurisy. Acute gastritis
7. Bronchial asthma. Bouts
8. Inhaled foreign body, laryngeal spasm, laryngeal oedema.
9. Hysterical.
10. Acidosis.

- Most common cause of laryngeal edema is (angioneurotic)
- Most common cause of laryngeal spasm (tetany)

CHEST EXAMINATION

INSPECTION

1. Shape:

1. Symmetrical:

- Barrel chest:

B: bulge : B: ↑ AP diameter

emphysema / B.A.

2. Asymmetrical:

- Unilateral bulge:

- Unilateral retraction:

pleural effusion,
lung collapse,

pneumothorax,
lung fibrosis.

creates -ve pressure
تسحب

fibrous bands
نسيج

للتفريق يأخذ نفس
إلى وأغص هو بارصة
داخل
bulge retracts

2. Respiratory movements:

In different chest diseases, respiratory movements are limited over the affected side.



space increases
-ve Intra pleural pressure

المزج
مركزي
أضيق

mediast. shift
to other side
mediast. shift
to same side

تقلل الحركة
في الصدر
تقلل الحركة
في الصدر
pleura

PALPATION

1. Trachea:

- To assess whether the trachea is central or shifted to one side:

- Shift to the same side of the disease:

occurs in the same causes of unilateral retraction.

collapse
fibrosis

- Shift to the opposite side of the disease:

occurs in the same causes of unilateral bulge.

pneumothorax
pleural effusion
or both

2. Tactile Vocal Fremitus (TVF):

- Causes of increased TVF:

- Consolidation, e.g. pneumonia
- Cavitation, e.g. lung abscess
- Cavitation surrounded by consolidation, e.g. bronchiectasis.

- Causes of decreased TVF:

- Almost all other chest diseases.

تصلب ودمع
دمع
Consolidation

Consolidation

normal aerated lung
by - infl. cells
- fluid

becomes replaced

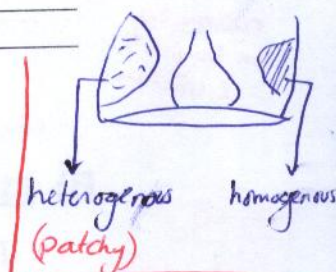
White Knight Love

PERCUSSION

Percussion notes in the chest

Note	Disease
Resonance	Normal
Hyper-resonance	Bilat - Hyperinflation as in asthma & emphysema = COPD unilat - Pneumothorax - Very thin people
Tympanitic resonance	- Tension pneumothorax
Dullness as liver	- Consolidation — solid - Cavitation — pus - Collapse — لا هواء - Fibrosis — rigid bands
Stony dullness as thigh	- Pleural effusion

= Flat dullness — ختم (بالوش صوت)



Causes of Dullness over the Base of the Right lung:

1. Supra-diaphragmatic causes:

- Pleural disease: pleural effusion, hydropneumothorax.
- Basal lung disease: Basal consolidation, Basal cavitation, Basal collapse, Basal fibrosis, bronchiectasis & Bilateral
- Heart disease: huge pericardial effusion.

2. Diaphragmatic paralysis.

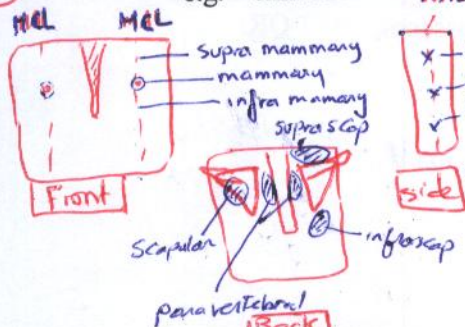
dullness ← rigid abd. liver

3. Infra-diaphragmatic causes:

- liver upper b level at 5 MCL
- Acute liver abscess.
 - Subphrenic abscess.
 - Marked ascites.
 - Pregnancy.
 - Huge abdominal mass.

4. Chest wall diseases:

e.g. tumours.



Supra infra

deep take insp

D → R
infra

D → D
supra

NB: — examinatio

of lt lung

rt lung

Back = L. Lobe

Ant = U. Lobe

Back = L. Lobe

Ant. — above 4th → U. Lobe

below 4th → L. Lobe

Bronchophony أقول auscult لازم في ↑TVF أعلى الصدر *
 wheezes أقول auscult لازم في ↓ الصدر * 12

AUSCULTATION

I. BREATH SOUNDS

A) Types

1. Normal Vesicular Breathing:

- It is heard normally in: NORMAL persons.

2. Vesicular Breathing with prolonged expiration:

- It is characterized by: prolonged expiration.
- It is heard in: BRONCHIAL NARROWING.

3. Bronchial Breathing: سرف

- It is characterized by: hollow sound (due to good conduction).
- It is heard in: CONSOLIDATION, CAVITATION. (as ↑TVF)
- It is confirmed by: bronchophony, aegophony, whispering pectorilquy.

normally
on larynx
& trachea

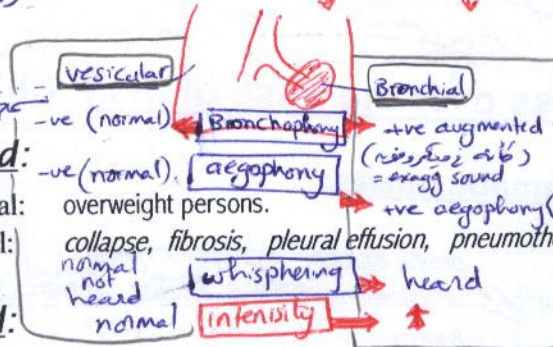
B) Intensity

1. Decreased:

- Physiological: overweight persons.
- Pathological: collapse, fibrosis, pleural effusion, pneumothorax, emphysema.

2. Increased:

- Physiological: very thin chest wall.
- Pathological: causes of bronchial breathing.



II. ADDED SOUNDS

A) Crepitations

(Crackles or Rales)

• Definition:

- These are interrupted (non-continuous) sounds produced by:
 - o Passage of air through fluid in the respiratory passages, OR:
 - o Pulmonary fibrosis.

alveoli
Fibrosis rigid wall

• Types:

a) Fine:

- Character: like the sound of rolling a tuft of your hair between your fingers near your ear.
- Heard in: **LVF** (basal & bilateral), **TB** (apical), **Early Pneumonia**.

b) Medium-sized:

"Consonating or Non-consonating"

- Character: like the sound of a crack.
- Heard in: **Bronchiectasis**, **Lung abscess**, **Pneumonia**, **Fibrosis**.

c) Coarse:

- Character: like the sound of loud gurgling & bubbling.
- Heard in: **Bronchiectasis**, **Lung abscess**, **Pulmonary oedema**.

طرق
1. clear
2. ringing
3. metallic sound
وهذا يحدث في
طرق ما يكون فيه
medium
موجلي جيد للصوت
Consolidating
Consonating

• Timing:

- They are usually: **INSPIRATORY**.

• Site:

- Localized: e.g. simple secretion, **FB**, tumour.
- Generalized: e.g. pulmonary oedema.
- Bilateral & Basal: e.g. **LVF** (fine), bronchiectasis (medium or coarse).
- Kronig's isthmus: e.g. **TB fibrosis** & cavity & Friedlander's pneumonia

inflamm.

surrounded by inflamm

• Persistence after cough:

- If you hear crepitations, ask the patient to cough & then auscultate again:
 - If sounds persist = organic pathological lesion. **Tumour**
 - If sounds dissappear: = simple secretion. **F.B.**

Clinical

crepitation
medium sized
inspiratory
Generalized

& ... after cough

B) Wheezes

(Rhonchi)

• Definition:

- These are continuous sounds produced by:
 - o Passage of air through the narrow bronchi (e.g. COPD, Bronchial asthma).

• Types:

- a) Sibilant: high pitched due to narrowing of **SMALL** bronchi.
- b) Sonorous: low pitched due to narrowing of **BIG** bronchi.

frequency

as snoring

Clinical

generalized Rhonchi
polyphonic wheezes

Sibilant Sonorous

• Timing:

- They are usually: EXPIRATORY.
- In severe obstruction: they may be heard also INSPIRATORY.

• Site:

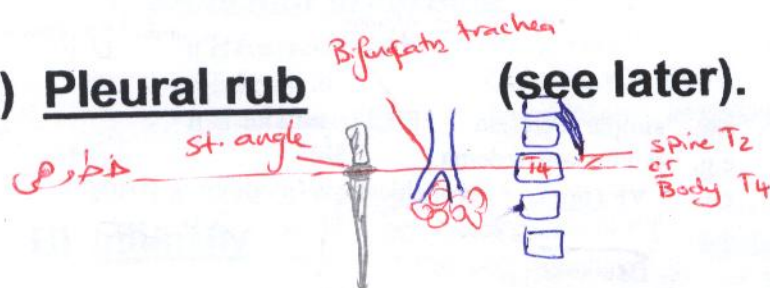
- Localized: e.g. simple secretion, FB, tumour.
- Generalized: e.g. COPD, bronchial asthma.
- Kronig's isthmus: e.g. Pancoast tumour.

• Persistence after cough:

- If you hear wheezes, ask the patient to cough & then auscultate again:

- iii. If sounds persist = organic pathological lesion.
- iv. If sounds disappear: = simple secretion.

C) Pleural rub



Despign sign

- Normally: on the back you hear BB till the 2nd dorsal spine in adults.
- + ve sign: on the back you hear BB below the 2nd dorsal spine in adults in the presence of enlarged mediastinal lymph nodes (they act as good conducting medium).
- Confirm by: vocal resonance (bronchophony) & whispering pectorliquy.
- Value: it indicates the presence of enlarged mediastinal lymph nodes.

↑ LNS ← +ve Despign
Bronchophony & whispering ← معرفت صوتی از ریه
از پشت! زای

Causes

1. T.B.
2. Bronchogenic Car.
3. malignancy

lymphoma leukemia

4. Sarcoid

- 1 Resp movt → ↓
- 2 ↓ TVF → ↓
- 3 ↑ TVF only ← Consolidation, Cantata, pneumonia, thorax and
- 4 only 2 Hyper R ← emphysema

Local signs of chest disease

5 to diff collapse & fibrosis
-1 Percussion -2 auscult

Chest disease	Inspection		Palpation		Percussion	Auscultation	
	Shape	Movement	Trachea	TVF		Breath sounds	Adventitious sounds
Curis Pural Effusion Pneumothorax Hydropneumothorax	Normal Bulge Bulge Bulge	Decreased Decreased Decreased Decreased	Central Opposite side Opposite side Opposite side	Normal Decreased Decreased Decreased	Normal - tender - Basal stony dullness Hyper-resonance (unilat) Shifting dullness	Decreased Decreased Decreased Decreased	Rub * ----- ----- -----
Consolidation Pneumonia Collapse Pneumosis	Normal Normal Retraction Retraction	Decreased Decreased Decreased Decreased	Central Central Same side Same side	* Increased * Increased Decreased Decreased	Dullness Dullness Dullness May be patchy dullness	Bronchial Bronchial Decreased Decreased	Crepitations Crepitations ----- Crepitations
Emphysema	Barrel	Decreased	Central	Decreased	Hyperresonance (Bilat) bronchitis	Decreased Vesicular, Prolonged expiration	Rhonchi, Crepitations
Chronic Bronchitis	Normal	Decreased	Central	Decreased	Normal	Vesicular, Prolonged expiration	Rhonchi

6 Syndrome of multiple negatives
Oral

1. Pl. effusion
2. collapse
3. fibrosis

7 any TVF → Bronchial Breatly
+ Bronchophony, Aegophony & ↑ intensity
whispering

8 Pneumonia is the only disease in 3 types of crepitation
Fine medium coarse } acc. to severity

↓ mvt, ↓ TVF, ↓ Percussion note, ↓ Breath sounds

9 To diff Consolidation & Cantata
not by examination but H/O
investing

10 any prolonged vesicular → wheezes

DISEASES OF THE PLEURA

1. **Dry pleurisy:** INFLAMMATION ONLY of the pleura.
2. **Pleural effusion (Hydrothorax):** FLUID in the pleura.
3. **Pneumothorax:** AIR in the pleura.
4. **Hydropneumothorax:** FLUID & AIR in the pleura.
5. **Fibrothorax:** FIBROSIS of the pleura.
6. **Mesothelioma (rare):** TUMOUR of the pleura.

DRY PLEURISY

ETIOLOGY

A) Primary Pleurisy:

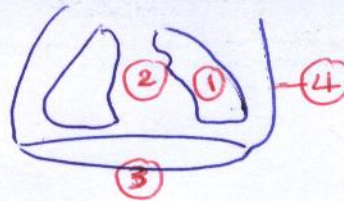
as dry pericarditis *جفاف الصفاق*

1. **Idiopathic** (probably viral: Coxsackie B or Echo virus): the most common cause
 - o Name: BORNHOLM DISEASE, or PLEURODYNIA.
 - o Age: Children & young adults.
 - o CP: Onset (Acute + fever), Duration (few days), Course (Recurrent).
2. **Infections:** *Viral, Bacterial, TB.* *infect*
3. **Inflammation:** *FMF.*
4. **Immunological:** *SLE, RA.*
5. **Iatrogenic:** *INH, Hydralazine, Procainamide.*
6. **Irradiation.** *Lupus like*
7. **Renal failure.**
8. **Rheumatic.**
9. **Post-traumatic.**
10. **Post-cardiotomy syndrome.**
11. **Myocardial infarction.** *late comp. cardi*
12. **Malignant infiltration.**

... Coxsackie
 - dry pericarditis
 - DCM

 - DM I

 - Meningitis



B) Secondary Pleurisy: (due to extension from surrounding structures)

1) Lung disease:

- INFECTIONS (TB, Pneumonia, Lung abscess), MALIGNANCY (Bronchogenic carcinoma) *by infiltration*

2) Mediastinal disease:

- INFLAMMATION (Pericarditis), MALIGNANCY (Lymphoma).

3) Subdiaphragmatic:

- ABSCCESS: *associated pleurisy* Amoebic liver abscess or Subphrenic abscess.

4) Chest wall disease:

- OSTEOMYELITIS: Sternum, Ribs or Vertebrae.

هذه
السبب
في
pl.
effusion

CLINICAL PICTURE

I Symptoms

1. Symptoms of the CAUSE.

2. GENERAL symptoms:

- Fever, malaise, headache, Anorexia fatigue

3. LOCAL symptoms:

A) PLEURITIC CHEST PAIN (due to irritation of the parietal pleura)

Pericarditis
1 S S cm 1 S
2 Retrost
shoulders

- Character & Duration: Severe Stitching, Continuous.
- Site & Reference: usually localized BUT may radiate:

1. In inflammation of the central part of the diaphragmatic pleura:
pain radiates to the shoulder through the phrenic nerve.

2. In inflammation of the outer part of the pleura:
pain radiates to the upper abdomen through the lower intercostals nerves.
(this may simulate acute abdomen).

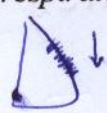
Middle
mediast.
(1) heart
(2) Trachea
& 2 Bronchi
(3) Phr. Nerve

- Increases by: Inspiration, cough & movements of chest wall.
- Decreases by: Holding breath & compressing the affected side.
- Associated symptoms: May be pain of dry pericarditis.

Also
• R.F
(AB Pericarditis)
• AMI
(inferior)

B) DRY COUGH due to irritation of the pleura. *dry* (except if Broncho pleural fistula & empyema)

C) DYSPNEA: due to ↓↓ of respiratory movements or to underlying lung disease.



2 diseases in chest
- lies on diseased site
1 pleurisy
2 White Knight Love

Signs

1. Signs of the CAUSE.

2. GENERAL signs:

e.g.

- Fever.

3. LOCAL signs:

Inspection

- Shape: ✓ normal.
- Movement: ✓ decreased on the affected side.

Palpation

- Trachea: ✓ central.
- TVF: ✓ normal.
- - May be palpable pleural rub. → لوعالی

as S₁ ↑ → stoppy
S₂ ↑ → Diastolic shock
murmur ↑ → thrill

Percussion

- ✓ Normal note.
- - Tenderness: on the painful site.

Auscultation

- Breath sounds: ✓ normal vesicular. ↓ intensity
- Additional sounds: ✓ "PLEURAL RUB"
- It is due to friction between the 2 layers of the rough inflamed pleura.
 - Site: Localised (at the painful site).
 - Character: Loud, Leathery, Low – pitched.
 - Increases by: with deep breathing & by pressing the stethoscope against chest the wall.
 - Disappears: on holding breath.

as Pain

Complications

- Pleural effusion. (exudate)

INVESTIGATIONS

- They have a negligible role in diagnosis, as dry pleurisy is diagnosed essentially: clinically

Pleuritic pain

+

Pleural rub

ام برب

- Investigations of the CAUSE.

Pericarditis

- 1 Pain
- 2 Rub
- 3 elev. ST

DIFFERENTIAL DIAGNOSIS

1. From other causes of:

acute chest pain.

ام برب

2. DD of:

the causes of dry pleurisy.

ام برب

طلة لانة من برب كبر

3. DD of

Medical Causes of Acute Abdomen

TREATMENT

Symptomatic:

- Analgesics & Anti-inflammatory drugs for the pain.

Specific:

- Treatment of the cause.

R.F: dialysis

if R.F

PLEURAL EFFUSION

DEFINITION

Accumulation of: fluid in the pleural space:

- Detected radiologically: if more than 300 ml. *not detected if < 300 by simple plain CXR*
- Detected clinically: if more than 500 ml. *C.T. can detect v. minimal amount*

ETIOLOGY

1. Serous effusion (ACCUMULATION OF EXUDATE)

A) Primary pleural disease:

It occurs in all conditions that cause dry pleurisy especially:

- TB:
- Malignancy.

"the most common cause of pleural effusion"

B) Secondary to:

- Lung disease.
- Mediastinal disease.
- Subdiaphragmatic.
- Chest wall disease.

See before

2. Pyothorax

(ACCUMULATION OF PUS = EMPYEMA)

- Severe inflammation of the pleura due to: Infection by pyogenic organisms, e.g. Staph. & Strept.

3. Hydrothorax

(ACCUMULATION OF TRANSUDATE)

- Heart failure (RSHF & LSHE), pericardial effusion, CP.
- Hepatic failure. *but ascites before pl. eff. due to Portal HTN*
- Hypoproteinemia, *↓ oncotic Pr.* e.g. Nephrotic syndrome. *systemic congestions ↓ pleural effusion "generalized edema"*
- Hypothyroidism.
- MEIG'S SYNDROME (Ovarian tumour, ascites, right pleural effusion).
- SVC OBSTRUCTION. *Back Pr.*

① unknown

② ↑ caps permeability (due to damage of wall by MPS deposition)

4. Hemothorax

(ACCUMULATION OF BLOOD)

1. Trauma: tapping of effusion & fracture rib.
to relieve distress but may injure B.V. (Bv. & N.V. in lower part of rib)
لذلك لا نؤذي الطبقة السفلى من الأضلاع
of rib
2. TB.
3. Malignancy: "characteristics of malignant effusion"
 (1) Massive, (2) bloody.
 (3) Rapidly re-accumulates after aspiration, / contains malignant cells.
 (4) Associated with mediastinal shift to the same side (due to underlying collapse).
 If cause is Malignancy w obstructs Bronchus → Collapse
 here not here
4. Hemorrhagic blood diseases. (generalized Bleeding)

5. Chylothorax

(ACCUMULATION OF LYMPH)

- Obstruction: of thoracic duct.
- Rupture: of thoracic duct.

1. Milky white
2. ↑ lipid content
3. dissolve by ether
4. → orange by Sudan III



CLINICAL PICTURE

Symptoms

1. Symptoms of the cause:

e.g.

TB toxemia.

- ! Night sweats
- ! Night fever
- ! loss wt
- ! loss Appetite

2. Symptoms due to effusion:

A) Small effusion:

Asymptomatic.

B) Large effusion:

- Dull aching chest pain.
- Dyspnea. — mech. Compression of lung } as pleurisy but stitching
- Dry cough.

irritation → cough R.

no effusion = Broncho pleural fistula

WhiteKnightLove

Signs

1. Signs of the CAUSE:

2. LOCAL signs:

Inspection

- Shape: ✓ unilateral bulge in large effusion.
- Movement: ✓ decreased on the affected side. normally +ve
- - Littin's sign: negative. *litten sign: in v. thin & illuminated*

Palpation

- Trachea & mediastinum:

a) Shift to the opposite side:

- o Small effusion: no shift.
- o Moderate effusion: shift of the apex to the opposite side.
- o Large effusion: ✓ shift of the apex & trachea to the opposite side.

b) Shift to the same side:

- o Malignant effusion: due to underlying lung collapse.
- o Chronic effusion: due to pleural fibrosis (if inflammatory)

- TVF:

- ✓ decreased on the affected side.

Percussion

- Dullness:

- Basal stony dullness, with the upper level rising laterally to the axilla.
- ~~Grocco's triangle~~
- ~~Garland's triangle~~

- Resonance:

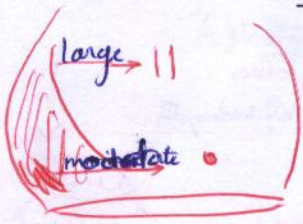
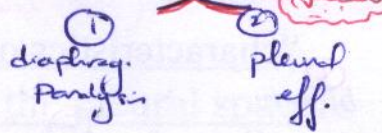
- ~~Skodiac resonance~~

Auscultation

- Breath sounds: diminished.
- Additional sounds: PLEURAL RUB may be heard.

oral

- Q - ? m. eff.
- pleural eff.
- Q - inspect
- uni Bulge, & mut, litten-u
- Q - litten-u
- diaphragm paralysis
- Q - ? Bronch. m. eff.
- e.g. TB. *Apical Cavities Fibrosis*
- We litten



COMPLICATIONS

1. Infection: empyema.

2. Chronicity: fibrosis or permanent lung collapse.

INVESTIGATIONS

I. IMAGING:

1. Chest x-ray:

a) Free effusion:

- Homogenous opacity obliterating the CP angle & rising laterally:

- Small effusion: small opacity with no shift of trachea or mediastinum.
- Moderate effusion: moderate opacity with shift of apex to opposite side.
- Large effusion: large opacity with shift of apex & trachea to opposite side.

b) Loculated effusion:

(especially in empyema)

- Fluid may be loculated due to adhesions.

- Loculated fluid may be either:

1) At the periphery of the lung

- Appears as a D-shaped capacity.

2) Within the fissures between the lobes (interlobar effusion).

- Appears as a rounded or oval opacity that may disappear with diuretics: phantom tumour or pseudo tumour.

2. CT chest:

- Sensitive in detecting small amounts of pleural effusion.
- May detect underlying lung disease e.g. malignancy.

II. OTHER INVESTIGATIONS FOR THE CAUSE:

- THORACOCENTESIS:

see later.

- Pleural biopsy:

e.g. for TB or malignancy.

- Sputum examination:

e.g. for TB or malignancy. only if

- Tuberculin test: good -ve

e.g. for TB.

- Thoracoscopy.

منظار وبيخ
Percutaneous

وتأخذ نسيج من

but
↓ sensitive

23

pleura

sputum

White Knight Love

THORACOCENTESIS:

diagnostic
Therapeutic
(Aspiration of effusion)

- Indications of aspiration:

A) Diagnostic: "May be needed for diagnosis of the cause by analysis of the pleural fluid"

a) Features of exudate & transudate effusions:

	TRANSUDATE	EXUDATE
<i>Proteins</i>	< 3 gm %	> 3 gm %
<i>Specific gravity</i>	< 1016	> 1016
<i>LDH</i>	< 200 IU / L	> 200 IU / L
<i>Cells (WBCs)</i>	< 1000 / cmm	> 1000 / cmm

NB Characteristics of TB effusion:

1. Exudate: rich in Lymphocytes & RBCs.

2. TB bacilli can be detected by:

- *Staining:* ZN stain.
- *Culture:* Lowenstein-Jensen medium or BACTEC.
- *Animal Innoculation:* Guinea pig.

b) Features of Pyothorax:

- The fluid is purulent & contains many pus cells.

c) Features of Hemothorax:

- The fluid is bloody & contains many RBCs.

d) Features of Chylothorax:

- The fluid is milky white & contains many fat, clears on addition of ether & stains orange with: Sudan III.

B) Therapeutic:

(see later).

DIFFERENTIAL DIAGNOSIS

1) From the syndrome of multiple negatives:

- Pleural effusion, collapse, fibrosis.

2) From basal lung lesions:

- Basal consolidation, collapse, fibrosis.

3) From subdiaphragmatic conditions:

- Amoebic liver abscess & subphrenic abscess.
- Tidal percussion may help in differentiation.

4) Differential diagnosis of the cause of effusion by:

- Main Features of the cause. → اشخ
- Nature of pleural fluid.

TREATMENT

1. Treatment of the cause:

e.g.

a) Tuberculous effusion:

- Anti-TB drugs. eg: INH, isoniazid (but not if Q: Tuberculous pl. eff. → درادنی)
- Corticosteroids: to ↓ inflammation & prevent fibrosis.

b) Malignant effusion: due to rapid accumulation

- Cytotoxic chemotherapy.
- Pleural sclerosis: by intrapleural injection of tetracycline. = pleurodesis

Indication of cortisone in TB.

2. THORACOCENTESIS:

(Aspiration of effusion)

INDICATIONS

- Massive effusion: { causing respiratory distress or reaching up to the second rib } ← Clinically: Percussion CXR
- Resistant effusion: not responding to treatment for 6 weeks.
- Effusion which becomes: secondary infected.

as if abscess
open drainage

سؤال
Thoracocentesis
1- diagnostic & therapeutic
2-

1. needle at oversurface of rib

PRECAUTIONS

- 2 • Aspiration should be done slowly.
- 3 • Aspirated amount should not exceed 1000 cc at a time.

[acute pulm. edema unilat]
sudden exp. of collapsed lung.

BENEFITS

1. Relieves dyspnea.
2. Reduces pleural fibrosis & lung collapse.
3. Reduces the intrapleural pressure, so the lymphatics will open and absorb the remaining amount of fluid. لأن الضغط في الجوف

COMPLICATIONS

1. Neurogenic Shock:

- Due to severe pain.

if injure nr. due to entry below rib

2. Acute pulmonary oedema:

- Due to sudden expansion of the collapsed lung.

3. Introduction of:

- Air → hydropneumothorax.
- Blood → hemothorax
- Infection pus → empyema.

PNEUMOTHORAX

27
 زنگنه
 جاست
 Case
 السته دي
 Case 25H

DEFINITION

Accumulation of: air in the pleural space.

ETIOLOGY

1. Spontaneous pneumothorax (non-traumatic):

تلقائي

a. Primary (benign) spontaneous pneumothorax:

○ Incidence: most common in males between 20 – 40 years especially smokers.

○ Cause: **UNKNOWN** (Spontaneous), there is no underlying disease:

It may be due to: unexplained rupture of a pulmonary bleb which is a localised collection of air most commonly in the apical zones.

It may be due to: a crack in a pleural scar resulting from a previous pneumonic patch.

It is spontaneously absorbed, but tends to be recurrent.

excellent prognosis

b. Secondary spontaneous pneumothorax:

○ Incidence: related to the underlying disease.

○ Cause: **KNOWN**, there is an underlying disease:

1. LUNG DISEASE:

○ Rupture of emphysematous bullae: → COPD.

○ Rupture of a TB cavity: → TB.

○ Rupture of a lung cyst or abscess.

- SEVERE ASTHMA. ; also rupture of emphysema bulla (same mech as COPD)

2. OESOPHAGEAL DISEASE:

e.g. tumour, (rare).

3. INFRADIAPHRAGMATIC DISEASE:

- Rupture of a subphrenic abscess.

pl. eff.

- ① Lung
- ② Mediast.
- ③ Subdiaphragm.
- ④ Chest wall

most common cause of pneumothorax
 rupture of bullae
 (Case COPD)
 rupture of bullae
 rupture of bullae
 rupture of bullae

هو = Pneumo
 هو = Hydro
 هو = Pneumo



هو = Pneumo
 هو = PyoPneumo

2. Traumatic pneumothorax:

a. Accidental pneumothorax:

- o Wounds: penetrating chest injury.
- o Invasive measures: e.g. surgery, thoracocentesis, pleural biopsy.
- o Endoscopy: bronchoscopy, oesophagoscopy.
- o MECHANICAL VENTILATION.

b. Artificial pneumothorax:

- o Collapse therapy previously used for treatment of TB.

Obsolete now

CLINICAL TYPES

1. Closed pneumothorax:

- The tear in the pleura is closed after the occurrence of pneumothorax.
- So the manifestations will:

2. Open pneumothorax:

- The tear remains open forming a fistula which communicates the pleura with the outside atmosphere.

- So the manifestations will:

3. TENSION PNEUMOTHORAX:

- The tear is valve-like allowing air to enter the pleural sac during inspiration but preventing its escape during expiration.
- So the manifestations will be:

CLINICAL PICTURE

Symptoms

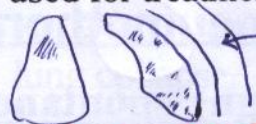
1. Symptoms of the cause:

e.g. COPD.

rupture of emphy. Bulla

IPPV
Intermittent
Positive
Pressure
ventilatory
pr. C_{AB}
C_{AB} = 20-30
cm H₂O

خروج هواء
Bronchus
& dies of feather
esoph.
Tension pneumo
Mediastinitis

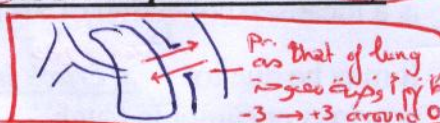


ناب TB و علاج او مثل قود
pleura
من يودي الى
Collapse lung
as to ↓ pathology
(palliative)
not effective

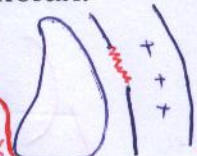
[3] Pr: -ve 4, 3, 2
↑ slightly

mostly dry type

reabsorption of air
subside spontaneously.



Pr. as that of lung
-3 → +3 around 0
cm H₂O



Normal Intrapleural
Pressure = -5 cm H₂O
this small amount of
air -5 → -4, -3, -2

normal pr.
changes inside
lung

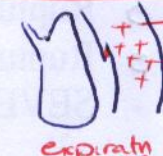
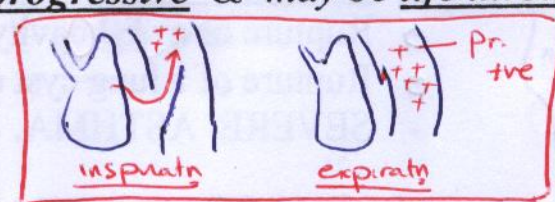
insp: -3
exp: +3 (elastic recoil)

Normal atm. pr. Zero cm H₂O

remain stationary.

Pr: around Zero

Zero الى 5
درج 5- الى



Pr: +ve

2. Symptoms due to pneumothorax:

A. In closed & open pneumothorax:

unilat 99%

cap/ caps in
pleura &
it's
pain
sensitive

- Chest pain: sudden, / severe, tearing, increasing with inspiration.
- Dyspnea: acute onset.
- Dry cough: acute onset.

① Pain → limitat

② ↑ intrapleural pr. → limitat lung compression
"Collapse"

B. In tension pneumothorax:

"4 manifestations"

- Acute chest pain. (sudden stretch of parietal pleura by huge amount of air)
- Dyspnea (severe) & cyanosis.
- Acute RVF. = Acute cor pulmonale
- Shock.

RVF
↓↓↓ COP
Signs

due to pH
↓↓ COP

Collapse

Central
sudden severe hypoxia
due to " " collapse

Sudden Severe Collapse → Sudden Severe hypoxia → Sudden Severe Pulm. VC → Sudden Severe pH. → Acute RVF

- Massive MI
- Massive PE
- T. Pneumo
- Massive collapse

1. Signs of the CAUSE:

e.g.

COPD.

emphysema
Ch. Bronchitis

① Hyperinflated chest
② wheezes

Subacute cor pulmonale
Minute Perforation
Barel
HyperR

2. LOCAL signs:

Inspection

- Shape: unilateral bulge in: tension type.
- Movement: decreased on the affected side.

closed & open
air (↓) سكتة
bulge جاس

Palpation

- Trachea & mediastinum: shifted to the opposite side in: tension type.
- TVF: decreased on the affected side.

Percussion

unilat ———→ Emphysema (Bilat)

- Hyper-resonance or tympanitic resonance. (tension type)

Auscultation

- Breath sounds: diminished. حمة هوا عذبة
- Additional sounds: amphoric breathing in: open & tension types.

pleural space ملا تأخذ نفسك الهواء فيس
ري، اصمعة

- How to detect level of diaphragm by palpation
- " " " obstructed air way by inspection

INVESTIGATIONS

1. Chest X-ray:

a. Characteristically, there is the line of pleura forming the lung edge:

B.r. vascular markings
- Outside this line: اسود جزء لونه في

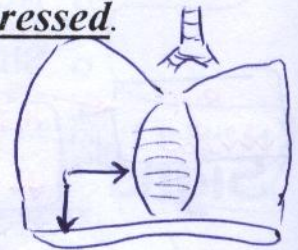
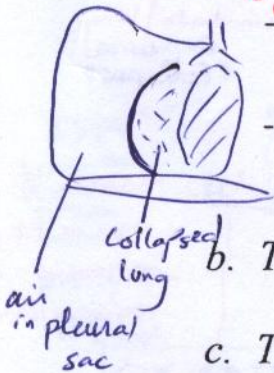
Homogenous jet-black translucency devoid of lung markings (pneumothorax).

- Inside this line:

The collapsed lung lies close to the mediastinum.

b. The trachea & mediastinum may be shifted to the opposite side.

c. The cupola of the diaphragm may be flattened or depressed.



2. Intrapleural manometry:

For recording the intrapleural pressure:

a) In closed pneumothorax: the pressure rises, but remains negative.

b) In open pneumothorax: the pressure oscillates around zero. -3 → +3

c) In tension pneumothorax: the pressure rapidly rises to become zero, and then becomes progressively positive.

3. ABG:

- Hypoxia. Collapsed lung

DIFFERENTIAL DIAGNOSIS

1. Differentiation of pneumothorax from:

- Other causes of: acute chest pain.

- Other causes of: acute dyspnea.

- EMPHYSEMA. Bilat. Hyperresonance / Pneumothorax unilat H.R

2. Differentiation of tension pneumothorax from:

- Other causes of: acute chest pain, dyspnea, acute RVF & Shock:

- Massive myocardial infarction. : typical ECG, chestpain, eng.
- Massive pulmonary embolism.
- Massive lung collapse.

3. Differentiation between closed, open & tension pneumothorax:

	Closed	Open	Tension
1. <i>Dyspnea</i>	mild	mild	severe
2. <i>Cyanosis</i>	absent	absent	usually present
3. <i>Shock</i>	absent	absent	usually present
4. <i>Acute HF</i> RVF	absent	absent	usually present
5. <i>Mediastinal shift</i>	absent	absent	usually present
6. <i>Amphoric breathing</i>	absent	present	usually present
7. <i>Intrapleural manometry</i>	-ve	See before around zero	+ve

TREATMENT

1. Conservative treatment:

- It is indicated in: primary spontaneous pneumothorax with:
 - Mild symptoms.
 - No increase in intrapleural pressure. ولو زاد يبقو بره -ve
 - Small pneumothorax (< 20 % of the hemithoracic volume). by CXR
- It consists of: bed rest & analgesics.
- Spontaneous absorption of air occurs allowing re-expansion of the collapsed lung.

2. Under water seal aspiration:

- It is indicated in:

- Severe symptoms.
- Severe increase in intrapleural pressure. Open Reaches zero
Tension Reaches +ve
- Large pneumothorax (> 20 % of the hemithoracic volume).
- Tension pneumothorax.

CXR
Manometer +ve

"pleuro desis"

3. Pleural sclerosis:

(production of pleural adhesions)

- It is indicated in: recurrent cases → by spontaneous
- It is done by intrapleural injection of: tetracycline.

4. Surgery:

(closure of the tear) إقفله جراحياً

- It is indicated in:

- Failure of aspiration (lung fails to expand within 10 days).
- Recurrent pneumothorax.
- Bilateral pneumothorax. (V.V.V. rare)

لو الجرح عاقلش في خلال يومين - ٥ أيام

و الرئة تغدو الجرح يقفل

و إذا اكمل انتظار راحة ١٠ أيام فلو لم يحدث

إقفله

لا ينفع أسوية عصبه وأسوية
حالة أدخل إقفله
و خلاص

5. Treatment of tension pneumothorax:

(medical emergency)

a) Life saving measure:

الطيان هيموت طانه صقناخ نكل نفس ناخو

- Immediate under water seal aspiration: to ↓ the intrapleural pressure rapidly.

b) Treatment of the 4 manifestations:

- Immediate relief of pain: pethidine 50 mg IM.
- Immediate relief of dyspnea: oxygen inhalation.
- Immediate ttt of heart failure. Diuretics, Digitalis
- Immediate ttt of shock. +ve inotropics

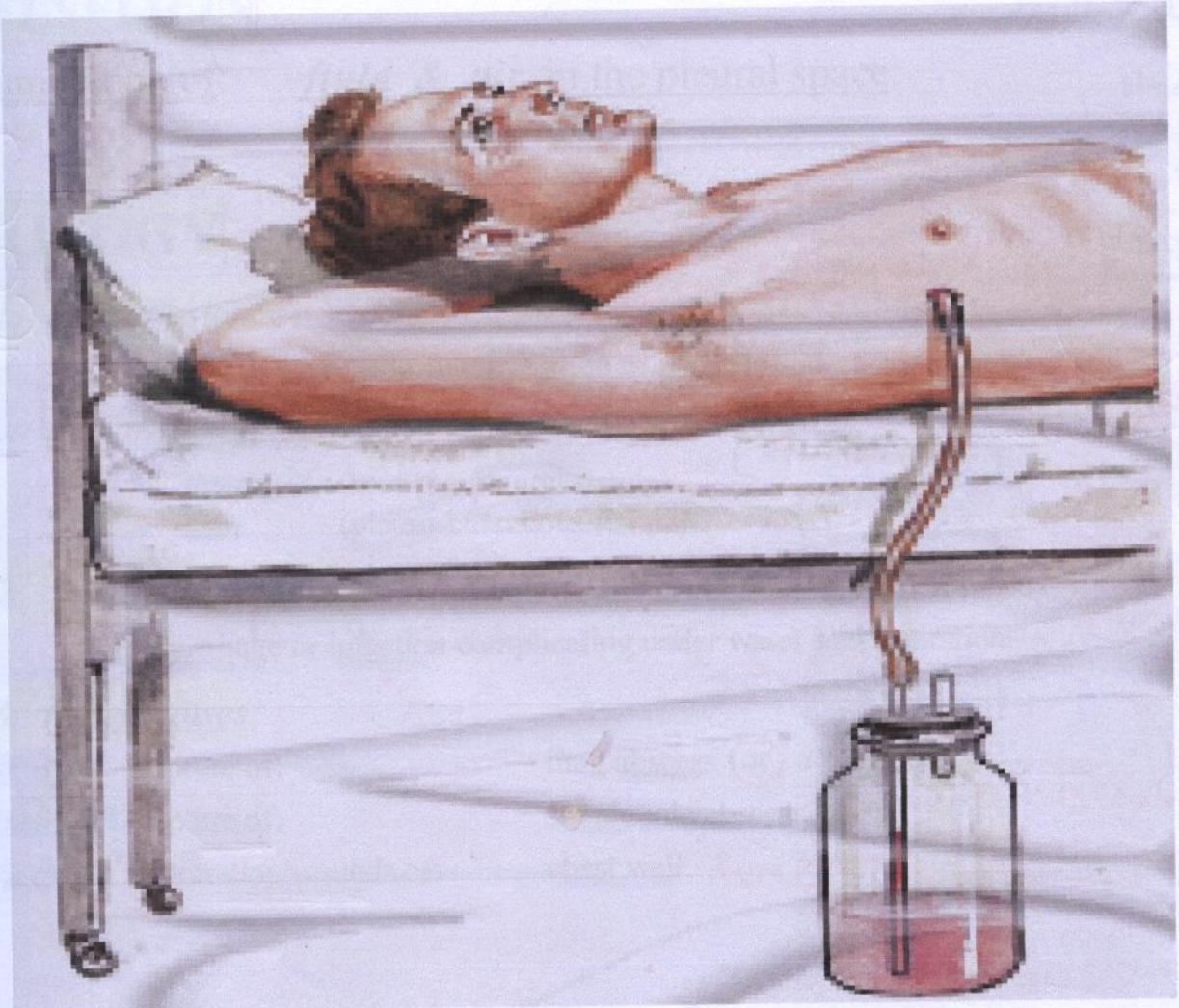
6. Treatment of the cause:

in secondary pneumothorax.

* يدخل أنبوب في الصدر (مؤبلة) في pleural sac الذي مليان به هو
 32 وهو داخله في انزلة من الساحة الأخرى تحت الماء

UNDER WATER SEAL air drainage

لأنه
 أنبوبة تحت الماء =



● during expnate → strain → ++ Intra pleural pr.

قوله مخزن ← نزول الهواء في الأنبوب ← bubbling in water إلى أن كله الهواء

الذي في pleural sac ← خلع وراح الانزلة → Q Lung expand

بح ينفذ & lung reexpand

1. Bubbling stops
2. Symp. improve
3. CXR

2-nd if fail → surgery

Q why under water → عتاه انكاس insp

Q why small tube?

عنه الهواء الذي جاء ليطلع مرة الانزلة

White Knight Love
 ساعه تسفط الماء لانها اقل

Hydro- , Hemo- & Pyo- PNEUMOTHORAX

1

DEFINITION

Accumulation of: fluid & air in the pleural space.

2

ETIOLOGY

1. Post effusion:

aspirals
fistula

- Introduction of air during aspiration of pleural fluid.

→ v. destructive

- Empyema opens into a:

- Bronchus (broncho-pleural fistula).
- Skin (pleuro-cutaneous fistula).

expectorate pus
Pyopneumo

(thoracocentesis
Complicatn)

Pus + air

Pus + air

2. Post-pneumothorax:

- Hemorrhage or infection complicating under water seal aspiration.

3. Other causes:

- Rupture of:

lung abscess or a TB cavity.

- Rupture of:

subphrenic abscess.

Trauma Penetrating wounds of:

chest wall. hemo pneumo.

3

CLINICAL PICTURE

Symptoms

- Chest symptoms: chest pain, dyspnea, dry cough.

- Symptoms of: suppurative lung syndrome in cases with bronchopleural fistula.

- Symptoms of: the cause. eg T.B.

- Night
- Night
- loss
- loss

repeated cough
+ ↑ amount of expectorata

- 1 - purulent
- 2 - feated
- 3 - related to Posture
- 4 - " " Seasonal

33

WhiteKnightLove

II Signs

1. Signs of the CAUSE:

e.g.

TB.
 Apical fibrosis
 Apical cavity

2. LOCAL signs:

Inspection

- Shape: ✓ unilateral bulge in: large hydropneumothorax.
- Movement: ✓ decreased on the affected side.

Palpation

- Trachea & mediastinum: ✓ shifted to the opposite side in: large hydropneumothorax.
- TVF: ✓ decreased on the affected side.

Percussion

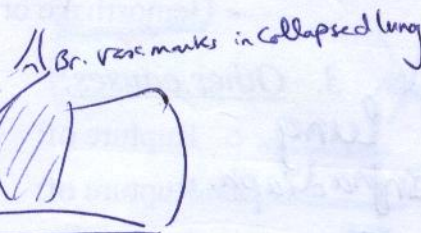
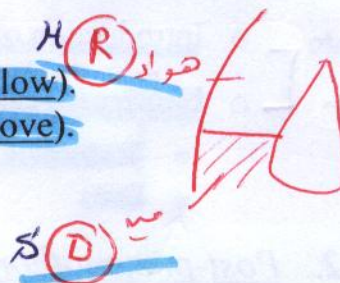
- D - Stony dullness with horizontal upper level
- R - Hyper-resonance or tympanitic resonance
- Shifting dullness: is characteristic.

(below)

(above)

Auscultation

- Breath sounds: ✓ diminished



INVESTIGATIONS

1) Chest x-ray:

a. Characteristically, there is the line of pleura forming the lung edge:

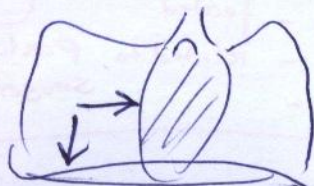
- Outside this line: ✓ ① ✓ ② Basal opacity ✓ ③
- Below: ✓ ④ homogeneous opacity obliterating the costophrenic angle, and has a horizontal upper level (air-fluid level).
- Above: homogeneous jet-black translucency devoid of lung markings.
- Inside this line:
 - The collapsed lung lies close to the mediastinum.

effusion ← Dull

← HR
Pneumo

b. The trachea & mediastinum may be shifted to the opposite side.

c. The cupula of the diaphragm may be flattened or depressed.



if Hydropneumothorax فلو

2) Aspiration of pleural fluid. →

اشرح كلاً
Theracocentesis
diagnostic

3) Methylene blue test:

- In the presence of broncho-pleural fistula, Methylene blue injected in the pleura will appear in the sputum.

لا تظهر الصبغة في سعال
sputum

5

TREATMENT

1. Treatment of the cause.
2. Aspiration of fluid & air using an under water seal.

PNEUMONIA

1

DEFINITION

also called pneumonitis

واحدة من أمراض الرئة، على الأطلاق
التهاب رئوي
hemophysis

1. INFLAMMATION of the lung:

(usually acute)

Chronic pn. is v. rare

acute > chronic
infect > non infect

LRTI

- Terminal airways.
- Alveoli.
- Interstitial tissue

infective & non-infective factors.

organisms

SLE (Abs against lung)

2. It may be caused by:

2

CLASSIFICATION

A. Classification by LOCATION (Anatomical):

1. Lobar pneumonia:

- Localised inflammation of:

unilat
homogenous

a single entire lobe.

2. Bronchopneumonia:

- Diffuse bilateral patchy inflammation of:

multiple lobules.

(affecting mainly: the alveoli adjacent to the bronchioles).

= lobular

3. Interstitial pneumonia:

- Inflammation of:

the interstitial tissue inbetween the alveoli.

v. rare

Sparring alveoli & Bronchi

less affected

more affected (adjacent)

B. Classification by CLINICAL PRESENTATION:

1. Acute pneumonia:

وهي بقاعة التي تحف لودماغ جسمك كوين

- It is the usual (common) type:

- Onset: acute.
- Duration: short (less than 3 weeks).
- Cause: the usual common organisms (see later).

مغير علاج

lobar : 1w
Broncho : 2w

2. Chronic pneumonia:

- It is the unusual (rare) type:

- Onset: gradual.
- Duration: long (more than 3 weeks).
- Cause: mycobacteria, fungi, non-infective.

SLE

TB

غير متبادر انه يجل
pn. في الجات اخرى
ولو عمل مبقه chronic

Oral opportunistic dty.

oral CMV effect on body

Oral Other Protzoa other than Pneumocystis carinii

- Entamoeba : Amoebic dysentery, colitis, liver abscess
- malaria Plasmodium
- Giardia
- Leishmania

oral فقد الشهية، الحمى، قلة الشهية
* VAP

GRF : Pneumonia in pt e stroke?

acute injury of Brain
due to vessels cause (disease)
جلو → hge
تجلو → thrombus

- Respirator {
1. Absent cough reflex (coma)
 2. Bulbar palsy : Δ tract is affected Bilat. (Pseudo)
 3. Orthostatic Pneumia (Bed prolonged recumbency)

Oral imm drugs.

oral Malignancy w ↓ imm : سرطان
lymphoma & leukemia

oral
Commonest Cause
of Neutrophils ↑↑ :
is Bacterial
infectn

Oral Types of WBCs < Eosino, Baso, Neutro & Monocytes, Lymphocytes

oral ↑ eosinophil < Allergy, Parasites

Oral hyposplenism

- ↓ Fc
1. Splenectomy
 2. auto : sickle cell : infarctn

Oral

Q. Etiology
 1- non-inf.
 2- infect.
 3- ppt factors

all systems ← CMV effect

opportunistic: attack ↓ imm.

1-TB
 2-CMV
 3-fungal
 4-pneumocystis carinii

38

C. Classification by ETIOLOGY:

I. INFECTIVE:

[Most common]

1. The infecting organism:

BACTERIA:

- Gm + ve cocci: **Strept. pneumoniae**, **Staph. aureus**, **St. pyogenes**
- Gm - ve bacilli: **H. influenzae**, **Klebsiella**, **Pseudomonas**, **Legionella**
- Anaerobes: by aspirate pneumonia, abscess
- Mycobacterium **TB**

Viruses: (non sp. skin rash)

- Influenza virus, **adenovirus**, **varicella**, **RSV**, **CMV**

Mycoplasma.

Chlamydia.

Rickettsia.

Fungi:

Protozoa:

Parasites:

atypical pneumonia

e.g. **Histoplasma**

e.g. **Pneumocystis carinii**

e.g. **Filaria**, **Toxoplasma**, **Ascaris**

e.g. **Candida**

e.g. **also I.E.**

2. The mode of infection:

- Community - acquired pneumonia:** Risk factors: overcrowding, cold weather

2. org. → **Strept. pneumoniae** (pneumococcal pneumonia): **most common**

- Others: **H. influenzae**, **Legionella**, **Mycoplasma**, **Chlamydia**, **Viruses**

- Hospital - acquired (Nosocomial) pneumonia:**

1. Risk factors: **Mechanical ventilators (VAP)**, **IV cannulae**

2. Organisms: ventilator acquired pneumonia

→ **Staph. aureus**: **most common**

- Others: **Klebsiella**, **Pseudomonas**, **Anaerobes**

- Aspiration pneumonia:**

1. Risk factors: **Stroke**, **Bulbar palsies**, **DCL**, **GORD**

2. Organisms: **in medulla**, **pharynx, larynx, palate, tongue**, **Disturbed Consciousness Level**

→ **Anaerobes**: **most common**

- Others: **Strept. pneumoniae**, **Staph. aureus**, **H. influenzae**, **Klebsiella**

- Pneumonia in the immunocompromised patient:**

1. Risk factors: **HIV**, **BM**, **advanced malignancy**, **immunosupp. drugs**

2. Organisms: **lymphoma**, **leukemia**, **Cortisol**, **imm. drugs**

→ **Pneumocystis carinii**: **most common**

- Others: **Fungi**, **CMV**, **TB**

T & B lymphocytes mainly T

cell mediated humoral

أكثر الأول

II. NON - INFECTIVE:

Least common

- Physical:
- Chemical:
- Immunological:
- Allergic:

radiation pneumonitis.

chemical toxins such as pesticides.

SLE

Abs against all tissue

R.F

other criteria

Löffler's syndrome (pulmonary eosinophilia).

① allergic Rx in lung in response to 1. drugs or 2. parasites

② characterized by inflamm. lung lesion

③ associated & heavy infiltrate of lung tissue by Eosinophils

④ peripheral blood eosinophilia

LOBAR PNEUMONIA

CAUSATIVE ORGANISM

It is usually caused by:

Strept. pneumoniae.

PREDISPOSING FACTORS

- AGE:
- PRECEDING INFECTION:
- ENVIRONMENT:
- ↓ IMMUNITY:

middle age.

upper respiratory tract viral infection.

cold weather,

overcrowding.

malnutrition,

hyposplenism.

↓ fn of spleen

2 PATHOLOGY

Consolidation is unilateral, usually involving one whole lobe.

The covering pleura shows acute pleurisy.

The disease passes through 3 stages over 7 days:

a) Stage of congestion:

- The alveoli are congested with:
- Air diminishes in the alveoli.

minimal exudate.

b) Stage of hepatization:

- The alveoli are filled with:

RBCs, WBCs, fibrin, thick exudates.

c) Stage of resolution:

- The exudate is liquefied by enzymatic activity & becomes copious & watery.
- The exudate is then cleared by:

Macrophages: through the blood and lymph vessels, or:

Cough mechanism.

not give cough sedative but give mucolytic & tussive

White Knight Love

- ① Malignancy
- ② Pneumonia
- ③ Mild hypoxia

العلی: عند دخول حالة chest: إدری تقول pale لأن كل عیاء chest بیقر عند anemia low hyperoxia ↓ BM + ↑ RBCs

CLINICAL PICTURE

Symptoms

A. General symptoms

- Fever:
 - Acute onset, high, associated with headache, malaise & rigors.
- Associated manifestations:
 - Somnolence & confusion: especially in the elderly. (Brain atrophy & atherosclerosis)
 - Headache & convulsions: especially in children.

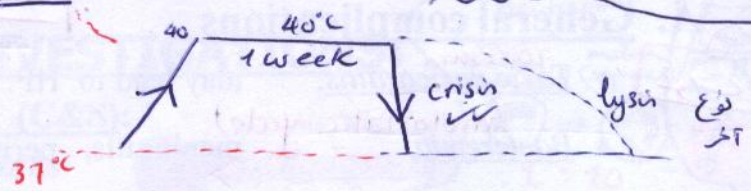
B. Chest symptoms

- Dyspnea. GRF: إتیفر
 - ① Pain
 - ② loss of aeration of part of lung
 - ③ inflamed lung → irritates lung → R.C. (Pulm. infarction)
- Pain:
 1. Pleuritic stitching.
 2. Upper abdominal pain & rigidity may occur due to diaphragmatic pleurisy & may simulate acute abdomen.
 3. if no pleurisy also pain of M. strain (cough)
- Cough:
 1. Dry: in the early stage. (minimal exudate)
 2. Productive: later:
 - With rusty sputum: during hepatization. (may be yellowish or greenish, with blood streaking).
 - With watery sputum: during resolution.

Signs

A. General signs

- Fever:
 - High continuous fever (40°C), lasts for about 7 days.
 - Resolves abruptly (by crisis).
- RR:
 - Tachypnea (shallow rapid respiration, 40/min).
- Face:
 - Pale & toxic.
- Lips:
 - Herpes simplex eruptions.



B. Chest signs (Signs of consolidation & pleurisy)

Inspection

- Shape: normal shape of the chest.
- Movements: limited on the affected side.

Palpation

- Trachea & mediastinum: central
- TVF: increased.

Percussion

- Dullness: on the affected area (lobe), due to consolidation.
- Tenderness: on the affected area (lobe), due to pleurisy.

Auscultation

- Breath sound: bronchial breathing.

↑ Intensity, Bronchophony, egophony, wheezing

- Additional sounds: crepitations:

Fine: early stage
Medium sized: during hepatization
Coarse: during resolution

- Pleural rub: is usually present. (Pleurisy)

طعق - Consonating
e - Consolidation

4 COMPLICATIONS

A. General complications

- Toxic myocarditis: may lead to HF. (Toxemia)
- Bacteremia: meningitis, peritonitis or suppurative arthritis. (metastatic infection)
- Septicemia: in severe cases may cause DIC, ARDS. due to marked permeability

B. Chest complications

- Unresolving pneumonia (persisting more than 2 weeks).
- Lung abscess. *staph Klebsiella*
- PLEURA: Pleural effusion, local spread, Empyema.

TNF-α is procoagulant → coagulation

Non Cardiac related pulmonary edema

UNRESOLVING PNEUMONIA

ربكيت مع
Complicatio
نظريه منفرد

- **DEFINITION:** Pneumonia that persists more than **2 weeks**.

- **CAUSES:**

1. Inadequate treatment.

2. Underlying lung disease e.g. **bronchiectasis** & **bronchial carcinoma**.

3. TB pneumonia. *if not give anti-TB*

4. Decreased immunity:

o **D**iabetes mellitus.

o **D**rugs: Corticosteroids & other immunosuppressives.

o **A**IDS. $\downarrow$ T & B $\downarrow$ & B11 depression

o **M**alignancies, especially lymphomas & leukemia

o **M**alnutrition.

*despite ttt -
ده هو الجفون الحف
لوحده غا اسبوع*

برقز العلاج



*Oral
dry infect on top of tumor*

*if cancer not removed
w types occurs*

Refactory H.f

Pn. resists unresolving

1) unresolving due to underlying cause (cancer)

2) recurrent at same site

*كل ما تحف بالادوية
تأني في نفس المكان
يقتر اليب هو
Cancer
في هذا المكان*

5

INVESTIGATIONS

I. IMAGING:

CXR

- **Homogenous** opacity: **unilateral**, usually involving **one whole lobe**.
- **Complication:** e.g. **pleural effusion**, **lung abscess**.

fibrosis (heterogenous)

II. LABORATORY INVESTIGATIONS:

1. **Sputum examination (C&S):**

- Pneumococci appear as: **Gm + ve diplococci**.

2. **Blood picture:**

- **Leukocytosis:** *neutrophilia (polymorphs)*

3. **Blood culture:**

- **Pneumococci:** *may be detected*

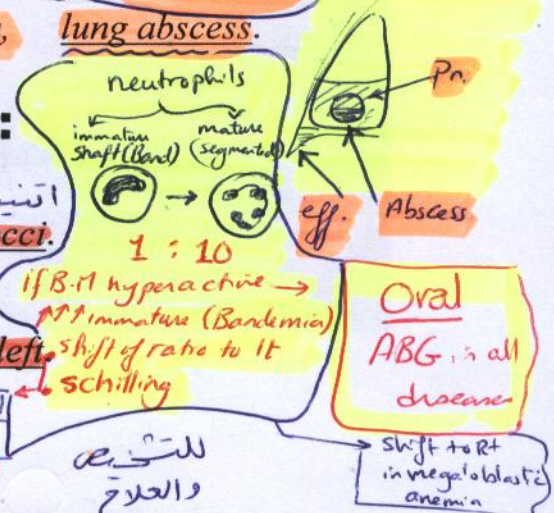
4. **ABG:**

- May show: **Hypoxia & Hypocapnia**.

5. INVESTIGATIONS FOR COMPLICATIONS:

- Culture of aspirated pleural fluid.

exudate



*مزارة
صورة دم
مزارة
غارات دم*

Types of diarrhea for p 47:

- 1- inflammatory: $\text{مليح} \rightarrow \text{ر} \rightarrow \text{diarrhea}$ eg: shigella
Bacillary dysentery
2. Motility: thyrotoxicosis
3. Osmotic: inside lumen (Osm. pr.) سكر , عسل
4. secretory: eg: toxin, hormone
irritates cells $\rightarrow$ secrete H₂O & electrolytes in lumen



Oral

Ciprofloxacin:

- Legionella
- UTI (ecoli)
- Typhoid
- Bacillary dysentery
- Spontaneous bacterial peritonitis

Oral P. 42

Causes erythema multiforme

- 1 atypical pn
- 2 viral (HSV)
- 3 drugs Hypersensitivity
 - penicillin
 - sulphonamides

erythema nodosum

= Steven Johnson Syndrome

Oral

infectious
RBCs

Abs

- 1 Malaria
- 2 Clostridium welchii

Oral

- Rf
- Post strept GN

- ITP
- Henoch

- Guillain Barre

- Type I DM

atypical pn. P. 43

- hemolytic anemia

Premature rupture of RBCs (before 120 ds)

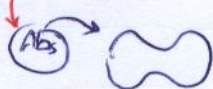
RBCs

extra RBC

- incompatible Bld transfusion
- Mechanical
- Hypersplenism
- Drugs

Autoimmune hemolytic anemia

as mycoplasma in some pts



agglutination
agglutination

Agg against RBCs
agglutination

Followed by

DIFFERENTIAL DIAGNOSIS

- Causes of: acute high fever with chest symptoms, e.g. acute lung abscess.

not considered
from multiple
negatives
as PTVF

- Consolidation should be differentiated from:
Collapse, fibrosis, pleural effusion.

dullness

Cavity & Consolidation
the same locally
diff by history & investigation

multiple negatives

- Differentiate between: lobar pneumonia & bronchopneumonia.

- FUO. (if unresolved)

TREATMENT

1. General Treatment:

- Bed rest.
- Proper nutrition. — malnutrition cause of

2. Specific Treatment: (antibiotics) ★ ★ ★

- Crystalline penicillin G: 1 million u / 6h IV (7 days).
- In allergy to penicillin:
 - Erythromycin: 500 mg / 6 h IV (7 days) or:
 - Cephalosporins.

strept.

- In resistant cases: antibiotics are given according to C & S.

from sputum

3. Symptomatic Treatment:

- Analgesics & Antipyretics.
- For early dry cough: cough sedatives, e.g. codeine phosphate.
- For productive cough: (not in resolution)
 - Mucolytics: e.g. Bromhexine.
 - Expectorants: e.g. Potassium iodide.
- Oxygen inhalation: in severe cases.

4. TREATMENT OF COMPLICATIONS:

- e.g. ttt of pleural effusion.

BRONCHOPNEUMONIA

CAUSATIVE ORGANISM



- BACTERIA:
 - Gm + ve cocci: Staph. aureus. *St. pyogenes*.
 - Gm - ve bacilli: *H. influenzae*, *Klebsiella*, *Pseudomonas*, *Legionella*.
 - Anaerobes.
 - Mycobacterium TB.
- Viruses:
 - Influenza virus, adenovirus, RCV, CMV.
- Mycoplasma.
- Chlamydia.
- Rickettsia.
- Fungi: e.g. Histoplasma, Candida.
- Protozoa: e.g. Pneumocystis carinii.
- Parasites: e.g. Filaria, Toxoplasma, Ascaris.

PREDISPOSING FACTORS

- AGE: extremes of age (children & elderly).
- UNDERLYING CHEST DISEASE: e.g. chronic bronchitis.
- ENVIRONMENT: cold weather, overcrowding.
- ↓↓ IMMUNITY: malnutrition, hyposplenism.
- HOSPITALIZED PATIENTS. : staph most common nosocomial of Bronchopneumonia

PATHOLOGY

A) LOCATION:

- Diffuse bilateral patchy inflammation of: multiple lobules.
(affecting mainly: the alveoli adjacent to the bronchioles).

B) LESION:

- Lobular consolidation & may be bronchiolitis & bronchitis.

lobular
"not lobar"

DIAGNOSIS

if Q Diagnosing Bronchopneumonia?

A: all as lobar pneumonia + جدول الفروقات

46

Similar to lobar pneumonia with the following differences:

	Lobar Pneumonia	Bronchopneumonia
Age	Middle	Extremes
Onset	Acute	More gradual
Offset	By crisis	By lysis
Duration	1 week	2 weeks
Pathology	Lobar consolidation	Lobular consolidation & bronchitis
Complications	Less common	More common as diffuse & staph (complicated)
Chest X-ray	Lobar consolidation uni homogenous opacity	Bilateral patchy opacities [fluffy cotton]

TREATMENT

- Same lines of treatment of Lobar pneumonia, **BUT:**
- Specific ttt (Antibiotics): depends on the causative organism (see later).

الفرد

Special types of pneumonias

1. BACTERIAL

Gm+ve cocci

a) Staphylococcal bronchopneumonia:

- Commonly occurs in: hospitalized patients.
- Commonly follows: a viral illness. *severe tissue breakdown*
- There is tendency to: ABSCCESS FORMATION *فرج* → empyema.
- May take a fatal course leading to death from Staph. septicemia.
- Treatment: Cloxacillin **or** Vancomycin. *DIC ARDS*

also in I.E.
if sensitive to penicillin

Gm+ve cocci

b) Streptococcal bronchopneumonia:

- Characteristically associated with: MARKED TOXEMIA.
- Treatment: Penicillin G **or** Erythromycin. *strept is? if sensitive to penicillin*

Gm+ve bacilli

c) Friedlander's pneumonia: (Klebsiella)

- Characteristically affects: APICAL ZONES (DD of apical lesions).
- There is tendency to: ABSCCESS FORMATION *فرج* → empyema.
- Characteristically there is: viscid, purulent, blood-stained sputum.
- Treatment: Aminoglycosides **or** Cephalosporins. *eg: gentamycin 3rd generation v-effective on -ve bacilli severe tissue breakdown*

also D.D.
- TB
- Pncostis
- Friedlander's

Gm+ve bacilli

d) Legionnaire's disease: (Legionella pneumophila)

- Occurs in: outbreaks **or** sporadically. *حالات فردية*
- Spreads usually in: places with contaminated cooling systems.

Oral

Multi-system Pneumonia?

- ① Legionella
- ② atypical *Mycoplasma Chlamydia Rickettsia*
- ③ non-infective *L.SLE*

Oral

most common cause of encephalitis

oral pr
Causes of death in legionella

- Acute Myocarditis
- Encephalitis
- ARF
- Hypotensive shock & electrolyte disturbance

- Clinical picture: [Multisystem affection]
 - General: gradual onset with fever & rigors.
 - Chest: cough (dry then productive) & dyspnea.
 - GIT: nausea, vomiting & watery diarrhea. *toxic effect on tubules only*
 - Heart: myocarditis. *toxic*
 - Neurological: mental confusion. *encephalitis (legionella is rare cause of encephalitis)*
 - Renal: ARF. *secretory diarrhea (due to toxins secreted)*
- Investigations:
 - CXR: Lobar consolidation **or** Bilateral patchy opacities.
 - Sputum: organism is detected with special stain: Gimenez.
 - Serology: Immunofluorescent antibody (IFA): ↑ Ab titre. *(inclined)*

Both are important
(ARF)

Treatment: Clarithromycin **or** Ciprofloxacin.

Macrolides eg: Azithromycin (Zithromax) *مجموعة*
erythromycin *oral* also used in H-Pylori
Quinolones: Broad spectrum *47 589*
Also in UTI Typhoid
White Knight love

2. NON - BACTERIAL

a) Viral pneumonia:

- 2nd most common (+) non sp. rash*
- The most common cause of pneumonia in: children.
 - Causative organisms: Influenza virus, adenovirus, varicella, RSV, CMV.
sp. to chest *synchial to imm*
 - Clinical picture: *rhinorrhea, sore throat*
 - General: Flu-like symptoms & may be skin rash.
 - Chest: dry cough, dyspnea, few physical signs.
 - Investigations:
 - CXR: may show bilateral patchy opacities.
 - Serology: antibodies to specific viruses.
 - Treatment: Antiviral drugs: e.g. Ribavirin for RSV.

Oral: also in Ht:

() Hepatitis C (Interferon)*

Other viruses only need symptomatic Ht

b) Atypical pneumonia:

- نظري*
- Causative organisms: MYCOPLASMA, chlamydia, rickettsiae.
it's name is called mycoplasma Pn.
 - Clinical picture: *atypical*

- Onset: **more gradual onset with a rather mild fever**

- Chest:

- Cough (*disabling*), dry, may be productive) & dyspnea.
- Minimal physical signs on the chest (*few crepitations*).

atypical **severe**
Extrapulmonary:

- General: fatigue, headache, myalgias.
- Skin: erythema multiforme.
- Blood: hemolytic anemia (*cold agglutinins*).
- Neurological: meningitis & encephalitis.

Investigations:

- CXR: Severe abnormalities on CXR despite minimal physical chest signs:

"Bilateral patchy opacities or GGA".

Treatment:

Clarithromycin

or Tetracyclin.

also Legionella

also pleuro des in pericarditis

marked discrepancy

48 between physical

White Knight Love

نظري
Case

لذلك اسير
walking Pn.
لأنه يقدر
يعيش وصرعه
لأنه حرارة
بيضا
فلا يلزم أنه
يقفل في سرير

erythema multiforme proximal
eryth. nodosum
skin tibia

lobar Pn.
dull → opacity
تحت كبر



Ground glass appearance

اسم قديم لـ atypical pneumonia

WALKING PNEUMONIA

- An old term that was previously used to describe:

- A clinically mild form of pneumonia. *fever*
- Affected patients do not need hospitalization.
- Causative organisms: MYCOPLASMA, chlamydia, rickettsiae.
(Atypical pneumonia)

c) Pneumocystis carinii pneumonia: (Protozoa)

- The most common opportunistic infection causing pneumonia in immunocompromised patients, especially: HIV.
- Diagnosis:
 - CXR: may show bilateral patchy opacities. *fuzzy cotton*
 - Detection of organism: Bronchoscopy with BAL or open lung biopsy. *as sputum analysis not sensitive*
- Treatment: Co-trimoxazole or Pentamidine. *لا تأخذ الـ BAL كاني*

ضعيف جدا
لذلك لا يهمل
الذي ليس عنده
مناعة
AIDS

Oral

BAL

Disease

- IPF
- Br. Carcinoma
- T.B.
- pneumocystis carinii

Sulfa
Oral لازم بيق حرق
نه حاجة زهنا لهوبس؟

adequate hydration

لازم يشرب
سوائل كثير
جدا لانه

هذا الدواء يعجل

crystals
in Renal tubules

قد يحول الى
renal stones

*لا تأخذ الـ
تقريباً*

Broncho
Alveolar
Lavage

ايمن Saline و ارجو و اعطاه

LUNG ABSCESS

Clinical
Long, short
نفس +

DEFINITION

- It is a localized suppuration in the lung with cavity formation and fluid level, that is not due to TB.

ETIOLOGY

1. Primary (inhalation) lung abscess:

the most common type

- 2 factors are responsible:

a) Inhalation of a specific material:

b) Absent cough reflex: eg

anaesthesia,

FB, vomitus.

prolonged convulsions.

- Causative organisms include:

☑ Anaerobes.

☑ Gm +ve:

Staph. aureus.

☑ Gm - ve:

Klebsiella.

H-influenza, strept. pneumoniae

epilepsy

coma

esp grand mal

case

epilepsy → inhalation

Abscess

anaerobes
+ 3 +ve Gm
+ 2 -ve Gm

as aspnath pneumonia

2. Secondary lung abscess:

A] Lung disease:

- Pneumonia, esp. *Staphylococcal & Friedlander's pneumonia.*
- Bronchiectasis.
- Bronchial carcinoma.
- Lung collapse.

Klebsiella

severe tissue break down

تكون الكيسة
Consolidation
في
cavity في

B] Mediastinal disease:

- Cancer oesophagus involving the lung.

C] Subdiaphragmatic disease:

- Amoebic liver abscess.
- Subphrenic abscess.

D] Chest wall disease:

- Penetrating wounds.
- Osteomyelitis of the sternum, ribs or vertebrae.

E] Pyaemia:

(pyaemic abscess)

- May occur due to infective endocarditis, osteomyelitis.
- The causative organism is usually Staph. aureus.

Multiple Bilat

في جميع أنحاء

في جميع أنحاء

في جميع أنحاء

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في جميع أنحاء

Pneumonia complicata (Abscess)

Bronchiectasis complicata (Abscess)

Abscess complicata (Bronchiectasis)

Collapse complicata (Abscess)

in IV drug addicts

PATHOLOGY

اذكر في الابدال
pyaemic
حتى لا تنسى

1. Primary (inhalation) lung abscess:

1. Site:

- Solitary, as inhaled F.B
- More common in the right lower lobe because the right bronchus is wider, and in direct continuity with the trachea.

2. Stage:

a) Pneumonic stage:

- There is an area of consolidation (pneumonic patch), and if it is superficial, the covering pleura will show acute pleurisy.

b) Stage of acute abscess:

- Tissue necrosis occurs in the consolidated area producing a cavity with a necrotic wall.
- The covering pleura shows acute pleurisy.

c) Stage of chronic abscess: (> 2M)

- The wall of the cavity becomes thickened by fibrosis.
- The covering pleura may show fibrous adhesions.

2. Pyaemic abscesses: (of 2M)

- They are usually: bilateral, multiple, small & of equal sizes.

CLINICAL PICTURE

1- Pneumonic stage:

2- Stage of acute abscess:

Symptoms

1- Acute high fever

After 1-2 weeks, the patient gets an attack of severe cough followed by expectoration of a huge amount of purulent foetid sputum.

This is followed by improvement of the general condition & drop of temperature.

The case continues as a suppurative syndrome in which there is paroxysmal cough related to posture with expectoration of excessive purulent foetid sputum when the patient lies on the healthy side.

Haemoptysis may occur.

Signs

- General signs: ↑ Fever, tachycardia & sweating (Toxaemia)
- Chest signs: Features of a cavity & may be pleurisy.

Pneumonia is except crepits only med coarse

if advanced fibrosis
→ retracts
& shift to same side

Clinical picture

Supp. Lung Syndrome

repeated attacks of cough & exp. of sputum

- Large
- Purulent
- Foetid
- related to posture (on healthy)

irritate by retained pus

as if drainage occurred

repeated attacks

as pus trickles to bronchus & irritates healthy bronchus & lung

3- Stage of chronic abscess: (Duration is > 2 months)

Symptoms

1 - Symptoms of suppurative syndrome.

2 - Attacks of acute condition on top of chronic (supp syndrome) retention syndrome:

The draining bronchus becomes obstructed with thick secretions.

So fever ris and sputum diminishes ↓

After few days the patient coughs repeatedly and expectorates the obstructing plug and symptoms of suppurative syndrome reappear.

3 - Progressive deterioration of general health.

Signs

General signs:

- Recurrent fever. As alternates between retention & suppurative
- Pallor, toxic face, loss of weight. anorexia (by toxemia) & catabolic (by toxemia)
- Puffiness of eye lids: due to chronic cough
- Clubbing of fingers: due to chronic toxemia

Chest signs:

"FEATURES OF A CAVITY & MAY BE FIBROSIS"

Inspection

- Shape: Normal but may be retraction due to excessive fibrosis.
- Movements: Limitation of movements on the affected side.

Palpation

- Trachea & mediastinum: Central but may be shifted to the same side due to excessive fibrosis.

- TVF: Increased.

Percussion

- Localized dullness over the site of the abscess.
- no pleurisy (not tender)

Auscultation

- Breath sound: Bronchial breathing, Bronchophony, aegophony & whispering pectoriloquy.
- Additional sounds: Crepitations; medium-sized or coarse.

Repeated attacks of cough & expectorates sputum

- large
- Pooled
- Purulent
- related to posture (↑ on healthy side)

ذلك لانه تساهل
by mucolytic & expectorant

supp. & retention
لانه حال يبدل بين
سائل صلب في صدره على طول

Anemia in chest
- infect
- malignancy

ACD
- infect
- inflamm
- malignancy

أسوأ حالة
toxemia
loss of just

Causes of clubbing
- chronic toxemia
- chronic hypoxia
- malignancy
toxemia
clubbing
hypoxia

Causes of puffiness eye lid

Oral & clinical

- Angioneurotic (Allergic) edema
- SVO
- Chronic cough - S/S - COPD
- nephrotic & nephritic
- hypothyroidism (HPS in kids)
- Blepharitis
- Cavernous sinus thrombosis

no fine only congestion

Oral diseases → 2ry amyloidosis

- Chronic infection eg: lung abscess, Bronchiectasis, T.B.
- " inflammation eg: SLE, RA, FMF

- Amyloid degeneration of kidney
- due to chronic toxemia
- leads to nephropathy
→ Nephrotic syndrome
→ Renal failure

COMPLICATIONS

A. General complications

- Toxemia: may lead to 2ry amyloidosis.
- Bacteremia: metastatic abscesses (e.g. brain).
- Anemia: < Toxic inhibition of BM
ACD

B. Chest complications

- Lung:
 - Pneumonia (spread of infection to other parts of the lung).
 - Fibrosis & bronchiectasis around the abscess cavity.
tracts on Bronchi
- PLEURA: Pleurisy, Pleural effusion,

Empyema

↓
frequent fibrosis
adhesions (compartments)
H: surgery

INVESTIGATIONS

1) Chest X-ray:

- Cavity with a fluid level.
- May show the cause: in secondary lung abscess.
eg: Br. Ca / lung collapse
- May show complications.
eg: pleural effusion

2) Culture & Sensitivity of the sputum.

3) CT chest:

- For accurate diagnosis & localization.

4) Bronchoscopy:

indicated to:

- Localize the site of the abscess
- Exclude the presence of underlying malignancy.
- Detect & remove foreign body.
- Drain pus & inject antibiotics into the cavity.

DIFFERENTIAL DIAGNOSIS

A. Causes of suppurative syndrome;

which is defined as:

"Cough with expectoration of excessive purulent foetid sputum, usually related to posture".

1. Lung abscess:

- onset
Symptoms → Cough
Signs, investigation
- 1 - Acute onset, no periodicity.
 - 2 - Expectoration increases on lying on the healthy side.
 - 3 - Signs are localized, & Chest X-ray reveals a cavity with a fluid level.

2. Bronchiectasis:

- onset
Symptoms → Cough
Signs, investigation
- 1 - Gradual onset, periodicity (more in winter, more in the morning).
 - 2 - Expectoration increases on leaning forwards.
 - 3 - Signs are bilateral & basal, & Chest X-ray reveals honey-combing.

3. Infected cystic lung: [Mucoviscidosis]

- Signs of bronchiectasis since birth or in early childhood.
- Chest X-ray reveals soap-bubble appearance or multiple fluid levels.

4. Empyema with broncho-pleural fistula: on top of pyopneumo

- Signs of pyopneumothorax.
- shifting dullness
- Methylene Blue test

B. Tuberculous cavity.

Apical
night
night
loss
loss

(+) Sputum : Bacilli

TREATMENT

1. Drainage of pus:

- 1) Physical therapy & Postural drainage.
- 2) Bronchoscopic aspiration & injection of antibiotics may be used.

2. Control of infection:

- Antibiotics according to sputum C & S.
- The most commonly used antibiotics are:

- ☑ For anaerobes: Clindamycin or Metronidazole.
- ☑ For Gm +ve, Staph. Aureus: Cloxacillin or Vancomycin.
- ☑ For Gm -ve, Klebsiella: Aminoglycosides or Cephalosporins.
- H. infl. : Ampicillin
- St. Pneumoniae: Penicillin or erythromycin

Oral uses of Metronidazole

- 1- Amoebiasis & giardiasis
- 2- Peptic ulcer LH-pylori
- 3- Crohn's (distal type) (anal part)
- 4- any anaerobes

IV - لا

3. Symptomatic Treatment:

- Analgesics & Antipyretics.
- For productive cough:
 - *Mucolytics:* e.g. Bromhexine.
 - *Expectorants:* e.g. Potassium iodide.

*or
pneumonia*

4. TREATMENT OF COMPLICATIONS:

- e.g. ttt of pleural effusion.

5. Surgery (segmentectomy or lobectomy) in:

- Chronic or huge abscess not responding to medical treatment.
- Severe recurrent hemoptysis. — *for hystic shock*
- Presence of underlying malignancy
- Complicating empyema or severe bronchiectasis.

Fibrothorax

as Both are irreversible

BRONCHIECTASIS

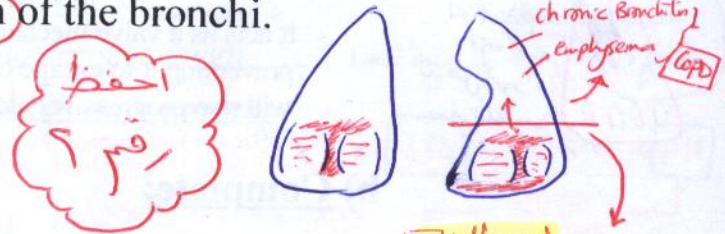
DEFINITION

Abnormal & permanent dilatation of the bronchi.

Clinical
Long & short
fresh +

PATHOLOGY

- Bilateral & Basal affection.
- Bronchi are inflamed, dilated, full of mucus & pus. The surrounding lung tissue may show consolidation & fibrosis.
- Bilateral emphysema of the upper parts of the lungs occur due to chronic bronchitis resulting from aspiration of infected sputum into the healthy bronchi.



- | | | |
|---|---------------|-----|
| 1 | inflamed | جوة |
| 2 | dilated | |
| 3 | mucus | برة |
| 4 | pus | |
| 5 | Consolidation | برة |
| 6 | Fibrosis | |

- 1- Spread of infection → ch. Bronchitis → emphysema
- 2- Compensatory emphysema

ETIOLOGY

Long
Acquired

1. Congenital bronchiectasis:

a) Isolated.

b) Part of a syndrome:

1) Immobile cilia syndrome:

- Bronchiectasis. ✓
- Sinusitis. ✓
- Male sterility. ↓ motility sperms

2) Kartagener's syndrome:

- Bronchiectasis. ✓
- Sinusitis. ✓
- Dextrocardia.

Oral

Bronchiectasis

يقع من
Bilat & Basal

- TB.

Bronchiectasis in TB

هنا فقط
Hemoptysis
Presenting

- TB

2. Acquired bronchiectasis:

A. Infection & Fibrosis:

- Recurrent infections lead to destruction & weakening of the musculo-elastic tissue in the wall of the bronchi resulting in their dilatation.

- In addition, peribronchial fibrosis pull on the bronchi from outside leading to further dilatation.

- The most common infections causing bronchiectasis are:

- Viruses: Adenovirus. (sp. to chest)
- Bacteria: Staph. aureus, Klebsiella, Anaerobes, H. influenzae, Strept. pneumoniae.
- Mycobacteria: TB.

if it is the cause of infection → Bronchiectasis (Apical)
" " " " " → Bronchiectasis (Sicca)
" " " " " → Bronchiectasis (Sicca)

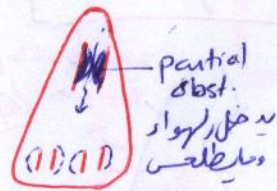
White Knight Love

NB: Bronchiectasis → apical COPD
 COPD → terminal (Peripheral airways) Bronchiectasis

B. Bronchial obstruction:

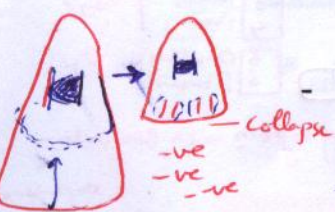
- Bronchial obstruction may be either:

a) Partial:



- It acts as a valve mechanism allowing air to enter during inspiration, but preventing it to escape during expiration, so the intra-bronchial pressure will rise progressively leading to bronchiectasis.

b) Complete:



- Collapse resulting from bronchial obstruction increases the negativity of the intrapleural pressure which exerts traction on the bronchi resulting in their dilatation.

- Bronchial obstruction may be due to either:

- In the lumen: FB or thick secretions.
- In the wall: Tumour.
- Pressure from outside: LN, tumour.

CLINICAL PICTURE

Symptoms

سعال مزمن

A. Cough & expectoration: (SUPPURATIVE LUNG SYNDROME)

- Persistent recurrent cough with expectoration of sputum which is:
 - Excessive
 - Mucopurulent
 - Foetid
 - Increases in the morning & on leaning forwards.
- Onset is gradual & the course is progressive over years with characteristic winter exacerbations.

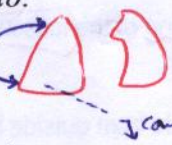
B. Hemoptysis:

HCO
 hemoptysis could be the only presentation of TB Bronchiectasis

- It may be blood-tinged or frank.
- It may be the only presenting symptom "bronchiectasis sicca hemorrhagica", esp. in cases secondary to TB.

C. Dyspnea:

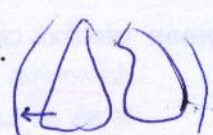
due to:
 Bronchiectasis → Fibrosis.
 Cause: COPD → Obstruction.



D. Chest Pain:

due to:

- Muscular pain because of severe cough.
- Pleuritic pain with occurrence of pleurisy.



E. Wheezing:

- Reflects extensive bronchiectasis.

apical: COPD + obstructed
 Basal: wide but full of pus (narrowing)

tissue damage of B.V.
 cough strain
 caseating destroy B.V. in wall of Bronchus
 frank Blood
 other sig: tissue damage of B.V. cough strain ruptures caps

Signs

1. General signs:

- Recurrent fever. *due to collaboration between retention on SLS + coughs drain then fever*
- Pallor, toxic face, loss of weight. *anorexia catabolic*
- Puffiness of eye lids: *due to chronic cough. lax eye lids*
- Clubbing of fingers: *due to chronic toxemia.*

- Oedema of lower limbs due to:

- 1- Cor pulmonale & RVF. *obstruction (apical) COPD Restriction (basal) fibrosis*
- 2- Renal amyloidosis. *chronic toxemia → amyloid degen. → nephrotic Syndr.*
- 3- Excessive loss of proteins in the sputum.
- 3- ↑ capillary permeability *due to ↓ O₂*

2. Chest signs:

- In the **basal parts**: there are signs of consolidation & may be fibrosis.
- In the **upper parts**: normal or signs of emphysema, Chronic Bronchitis.

Inspection

- Shape: Hyperinflated in **upper parts**. Retraction of the **basal parts** may be seen due to extensive **fibrosis**.
- Respiratory movements: Diminished, mainly in the basal parts.

Palpation

- Trachea & mediastinum: Central.
- T_{VF}: Increased on the **basal parts**. Decreased on the **upper parts**.

Percussion

- Patchy dullness: on the **basal parts**.
- May be hyper-resonance: on the **upper parts**. **COPD**

Auscultation

- Basal Parts:
 - Breath sound: areas of bronchial breathing.
 - Additional sounds: Medium-sized or coarse crepitations. *no fine*
- Upper parts:
 - Breath sound: Vesicular breathing with prolonged expiration. (**Bronchitis**)
 - Additional sounds: Rhonchi.

COMPLICATIONS

as abscess

E
E
E
+ PH
+ RF

A. General complications

- Toxemia: may lead to amyloidosis.
- Bacteremia: metastatic abscesses (e.g. brain).
- Anemia: $\leftarrow$ Tox. & BM ACO

B. Chest complications



- Lung:
 - Pneumonia (spread of infection to other parts of the lung).
 - Lung abscess (spread of infection to other parts of the lung).

- PLEURA: Pleurisy, Pleural effusion, Empyema.



- Pulmonary hypertension: $\rightarrow$ cor pulmonale & RVF.
- Respiratory failure: due to associated emphysema.

COPD
R.F. chest pain

INVESTIGATIONS

as abscess

+ Bronchography

1) Chest X-ray:

1. Basal parts:
 - Prominent cystic spaces having the characteristic: "Honey-comb appearance".
 - Opacities around the cysts due to consolidation.
2. Upper parts: "Hypertranslucency" due to emphysema.

2) Culture & sensitivity of the sputum.

- The most commonly isolated organisms are: H. influenzae & Strept. pneumoniae.

3) CT chest:

(the most important investigation)

- It provides excellent visualization of the bronchiectatic airways.
- It localizes the site & extent of bronchiectasis before surgery.

4) Bronchography:

- It also provides excellent visualization of the bronchiectatic airways.
- It shows the dilated bronchi as: cylindrical, saccular or fusiform.

5) Bronchoscopy:

- May reveal the cause in localized bronchiectasis.
- May be used for drainage of pus & injection of antibiotics.

site

✓ DIFFERENTIAL DIAGNOSIS

- Causes of suppurative lung syndrome. →
- (+) - Causes of hemoptysis. →

as abscess⁶⁰
اشرج كاملا
من
Abscess
+ hemoptysis

✓ TREATMENT

1. Drainage of pus:

- ✓ 3) Physical therapy & Postural drainage.
- ✓ 4) Bronchoscopic aspiration & injection of antibiotics may be used.

2. Control of infection:

- ✓ Antibiotics according to sputum C & S.
- ✓ The most commonly used antibiotics are:

☑ For anaerobes:	Clindamycin or Metronidazole.
☑ For Gm +ve, Staph. Aureus:	Cloxacillin or Vancomycin.
☑ For Gm - ve, Klebsiella:	Aminoglycosides or Cephalosporins.
☑ For H. influenzae:	Ampicillin.
☑ For Strept. pneumoniae:	Crystalline penicillin G. <i>new therapy.</i>

✓ 3. Symptomatic Treatment:

- Analgesics & Antipyretics.
- For productive cough:
 - ☐ Mucolytics: e.g. Bromhexine.
 - ☐ Expectorants: e.g. Potassium iodide.
- (+) • For airway obstruction: Bronchial obstr.
 - Bronchodilators.

✓ 4. TREATMENT OF COMPLICATIONS:

- e.g. ttt of pleural effusion.

5. Surgery (local resection or lobectomy) in:

- # CIP
 - Persistent or recurrent infection not responding to medical treatment.
 - Severe recurrent hemoptysis. → (for hagic shock)
 - Localised bronchiectasis.
 - Extensive Bilat.
 - ↓
 - lung transplant if Bilat.

CYSTIC FIBROSIS

(Mucoviscidosis)

DEFINITION

- A hereditary disease (autosomal recessive), affecting mucus-secreting exocrine glands & sweat glands. ^① - excessive production of mucus
- Most of the clinical features are secondary to obstruction by viscid mucus. ^② ^③ extensive cysts formation

CLINICAL PICTURE

1) Chest (Polycystic lung):

- Recurrent infections & bronchiectasis, usually in a young age.

2) GIT & Liver:

- Intestinal obstruction in the newly born (meconium ileus).
- Pancreatic insufficiency & malabsorption (fibrocystic pancreas).
- Jaundice & later cirrhosis (cystic liver).

3) Genitourinary:

- Male sterility (obliteration of the vas deferens).

Cong. Bilat absence of vas

INVESTIGATIONS

1) Chest x-ray:

- Soap-bubble appearance.
- Multiple fluid levels in cases of infection.

2) Pancreatic function tests:

- May be impaired.

3) Sodium in Sweat:

- Is markedly elevated (diagnostic).

TREATMENT

1- For polycystic lung: same as bronchiectasis.

2- For meconium ileus:

- Enemas & nasogastric suction.
- Surgery is rarely needed.

3- For pancreatic insufficiency:

- Pancreatic enzyme replacement.

لناي سي

Polycystic lung
infected cystic lung

كلى
cysts
pus في

++ sodium secretion in sweat

Supp. lung syndrome :- Birth

انسداد
int
الم
mucus

jaundice

malabsorp
(↓ exocrine
Pancreatic Enz)
- 2ry DM
(↓ endocrine fn)

oral clinical
Causes
of jaundice
:- chest
diseases

RESPIRATORY FUNCTIONS

1. Ventilation. تهوية
2. Perfusion. وصول الدم
3. Diffusion. تبادل الغازات

نظري
Discuss
Resp fn & Resp fn tests
سفری و کلینی

VENTILATION

DEFINITION

- It is the process of: movement of air in & out of the lungs.
- It is the process of: exchange of air between the lungs and the atmosphere.
- It includes: **INSPIRATION & EXPIRATION.**

PHYSIOLOGY

INSPIRATION (breathing in): is an **ACTIVE** process.

- It occurs when the muscles of the diaphragm and chest wall contract.
- The contraction of these muscles will:
 1. Expand the chest wall, the pleura, the lung & therefore will allow air to enter.
 2. Lowers the pressure inside the chest, & thus lowers the pressure in the airways:
 - Pressure gradient occurs from mouth to alveoli, so air rushes in.

EXPIRATION (breathing out): is a **PASSIVE** process.

- As the muscles relax, the elastic recoil of the lungs puts pressure on the gases inside.
- The pressure in the chest is now higher than the outside pressure:
 - Pressure gradient occurs from alveoli to mouth, so air rushes out.

Ventilation will depend on:

1. Diameter of the airways.
2. Compliance of:
3. Neuromuscular apparatus.

chest wall, pleura, lungs.

scleroderma # effusion fibrosis

Neuromuscular apparatus

I. RESPIRATORY CENTRE in the *medulla*; is controlled by:

A) Feedback from afferent receptors:

Sensors

A B C

1. AIRWAY RECEPTORS (Slowly adapting stretch receptors)

- Location: walls of the bronchi.
- Sensed by: expansion of the airways at the end of inspiration.
Stretch
- Function: send inhibitory impulses to **RC** through the vagus to terminate inspiration (Protective Hering-Breuer reflex).

2. BARORECEPTORS (Pressure receptors)

- Location: carotid sinus. (*also Aortic Arch*)
- Sensed by: changes in the arterial BP.
- Function: modulate ventilation according to those changes.

3. CHEMORECEPTORS

- Location: medulla (central), carotid arteries, aa (peripheral).
- Sensed by: changes in PCO_2 (central), PO_2 (peripheral), pH.
- Function: modulate ventilation according to those changes.

4. OTHER RECEPTORS (Proprioceptors)

- Location: muscles, tendons, joints.
- Sensed by: movement during exercise.
- Function: send stimulatory impulses to **RC**.

5- J- Receptor: sense pathological edema

B) Cerebral cortex:

- Provides some voluntary control of breathing.

II. NEURAL PATHWAY:

Conductors

- AHCs (Cervical & Thoracic segments of the spinal cord).
- Respiratory nerves (Phrenic & Intercostals nerves).
- NMJ.

III. RESPIRATORY MUSCLES.

Effectors

1000

سفر

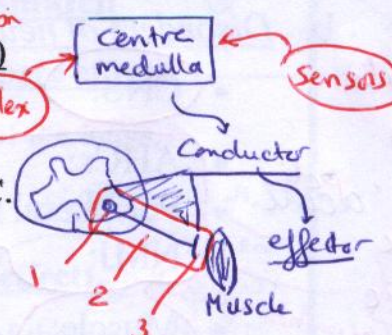
لأن
توسع
الرئتين
تفريح

hypotension $\rightarrow$ hypoxia $\rightarrow$ $\uparrow$ RR

hypertension $\rightarrow$ good perfusion $\rightarrow$ $\downarrow$ RR

central
peripheral

acidosis
 $\downarrow$
stimulatory
والفح
عسر



oral
 $\uparrow PCO_2$, $\downarrow PO_2$
stimulatory
والفح
عسر
202
exercice

HYPOVENTILATION

ETIOLOGY

Acute vs chronic

1 airway
2 chest, lung, pleura
3 N-M system

1. Obstructive hypoventilation: ($\downarrow$ diameter of airways)

a. Upper respiratory tract obstruction: e.g.

- Acute laryngeal oedema or inhaled FB. — (Acute)

b. Lower respiratory tract obstruction: e.g.

- Bronchial asthma. — Acute & chronic

- COPD. — chronic

angioneurotic

2. Restrictive hypoventilation:

a. Decreased compliance of:

- * chronic Lungs: ILD. = IPF = ILF
- * chronic, acute Pleura: Massive effusion, Tension pneumothorax.
- * chronic Chest wall: Chest deformities, / scleroderma / marked obesity (Pick synd).

Pressure (مقاومة انقباض)

Volume (مقدار انقباض)

$\downarrow$ compliance

Volume (مقدار انقباض) لا يتغير

b. Disorders of neuromuscular apparatus:

- CNS: — Center Head injury, CVS, Drugs (opiates & barbiturates).

- AHCs: MND, Poliomyelitis.

- Peripheral nerve: Polyneuropathy, e.g. GBS.

- NMJ: — conductor Myasthenia gravis.

- Muscle: — effector Myopathies.

↓

duchenne
dies of R.F.
due to effector
restriction

Guillan
Barre
acute post
infectious

سيفالوسپورين
pethidine
jans
morphine

MANIFESTATIONS

- Hypoxia.

اشبع
R.F.

- Hypercapnia.

اشبع
R.F.

IPF membrane surface area.
 emphysema ↓ PO₂
 Pulm. edema } V/Q ratio 66
 Pulm. emb }
 pneumonia }

DIFFUSION

DEFINITION

- It is the process of gas exchange between the alveolar air & the pulmonary capillary blood.
- It occurs through the alveolo - capillary membrane.

PHYSIOLOGY

- Diffusion will depend on:

1. Ventilation - perfusion matching:

- *Normally*, gas exchange depends on the proper matching of ventilation and perfusion, i.e. there should be equal distribution of the inspired air and perfusing blood in different parts of the lungs (V / Q ratio).

LV radioactive albumin

LV radioactive xenon

- *Abnormally*, gas exchange will become impaired if there is V / Q mismatch.

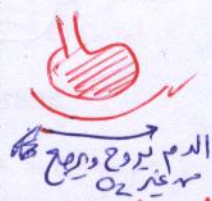
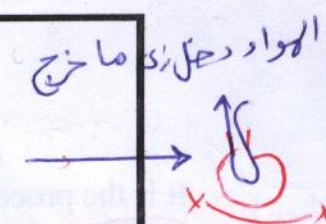
Causes of Ventilation - perfusion mismatch

a. Increased V / Q ratio: = Q ↓ (dead space effect)

- It means ventilation of unperfused areas of the lungs, e.g. *pulmonary embolism*.

b. Decreased V / Q ratio: = V ↓ (shunt effect)

- It means perfusion of unventilated areas of the lungs, e.g. *pneumonia*.

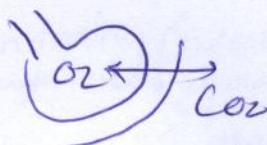


2. Surface area of diffusion.

3. Intact alveolo-capillary membrane.

4. Pressures of O₂ in the alveolar air & CO₂ in the capillary blood.

↓ IPF Pulm. edema



IMPAIRED DIFFUSION

ETIOLOGY

- ILD. *membrane*
- Emphysema. *S-A.*
- Pulmonary oedema. *↓ p_a*
- Pneumonia. *↑ v/p ratio*
Pulm. embolism

MANIFESTATIONS

- Hypoxia.
- Normocapnia.

PERFUSION

- It is the process by which the pulmonary circulation delivers blood to the gas exchange units so that: O₂ uptake & CO₂ elimination can occur.

= alveoli

v/p mismatch
in pulm. embolism

Respiratory Function Tests

I. Tests for specific pulmonary functions:

A. VENTILATION TESTS:

1. Respiratory volumes & capacities: (using the spirometer)

- See later.

2. Expiratory flow rates:

- Forced expiratory volume in the first second (**FEV₁**):
 - Normally: ~ 3800 ml.
 - Normally: $FEV_1 / FVC \sim 80\%$ (Timed vital capacity).
- Peak expiratory flow rate (**PEFR**).

These tests are impaired in: **OBSTRUCTIVE HYPOVENTILATION**

3. Lung compliance:

- The ratio of change in volume of the lung to change in intrapleural pressure.

4. Ventilation scan:

- Using radioactive Xenon (see pulmonary embolism).

B. DIFFUSION TESTS:

- Diffusing capacity: (CO transfer factor)

- It measures the quantity of **CO** that will diffuse in a time unit from the alveolar gas into the capillary blood.
- It is a useful measure for the integrity of the alveolo - capillary membrane.

C. PERFUSION TESTS:

1. Pulmonary vascular pressures.
2. Perfusion lung scan: using radioactive albumin (see pulmonary embolism).

II. Tests for general pulmonary functions:

○ ABG:

○ Pulse Oximetry.

ABG

① non invasive

② Continuous monitoring depends on light wave length

PO_2 & PCO_2

from femoral or radial

calculates absorbance of wave length of light

abs. ~ 6

absorbance of light

acc. to O_2 saturation
White Knight Love
displays

Tidal Volume:**TV**

- o It is the volume of air inspired & expired during: normal quiet breathing.
- o Normally ~ 500 ml.
- o Resting minute volume (RMV) = TV x RR (16 / min.) = 8000 ml / min.

أي قسم
- Co
- SV
- HR

Residual Volume:**RV**

- o It is the volume of air remaining in the lung: after maximum expiration.
- o Normally ~ 1200 ml.
- o It increases in: COPD. ++ Intra alv. pr. → septa broken → apl. Bullae

Forced Vital capacity:**FVC**

- o It is the volume of air that is maximally expired: after maximum inspiration.
- o Normally ~ 4800 ml (ERV + IC)
- o It decreases in: all cases of hypoventilation, especially restrictive hypoventilation.

Total Lung Capacity:**TLC**

- o It is the volume of air that the lungs contain when they are fully expanded.
- o Normally ~ 6000 ml (RV + FVC)
- o It decreases in: restrictive hypoventilation.

الأكسجين
الموجود
في جدران
القصبة
max insp.

f. obstr. → ↓ FVC (minimal)
↑ RV (مزيد) } ↑ TLC

هو بقدر
العصبات
فلو ضعفت

Expiratory Reserve Volume:**ERV**

- o It is the additional volume of air that can be exhaled after a normal tidal breath out.
- o Normally ~ 1200 ml.

ما أخزاه سوية بعد العظمى

Inspiratory Reserve Volume:**IRV**

- o It is the additional volume of air that can be inhaled after a normal tidal breath in.
- o Normally ~ 3100 ml.

له يخرجوا
تبقى الـ
فيخروجوا لو حركت

Functional Residual Capacity:**FRC**

- o It is the volume of air left in the lungs after a normal tidal breath out.
- o Normally ~ 2400 ml (RV + ERV)

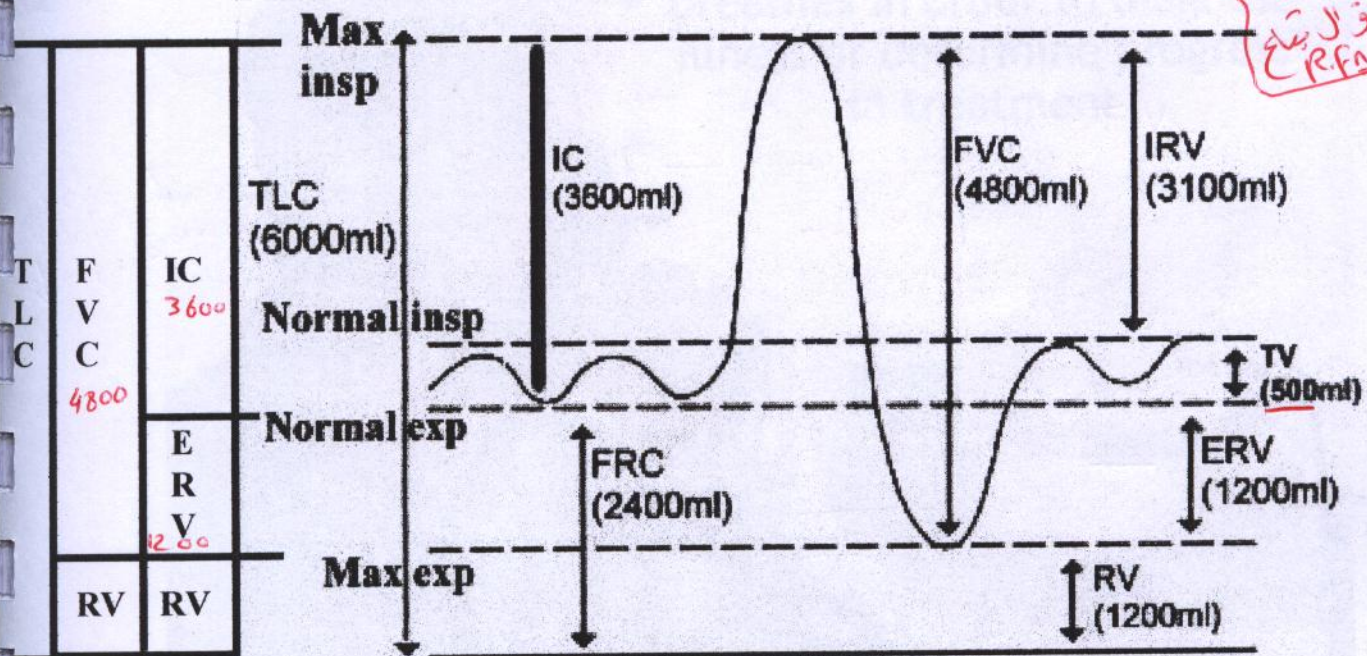
أكبر نفس بعد normal exp.

Inspiratory Capacity:**IC**

- o It is the maximum volume of air that can be inhaled after a normal tidal breath out.
- o Normally ~ 3600 ml (TV + IRV)

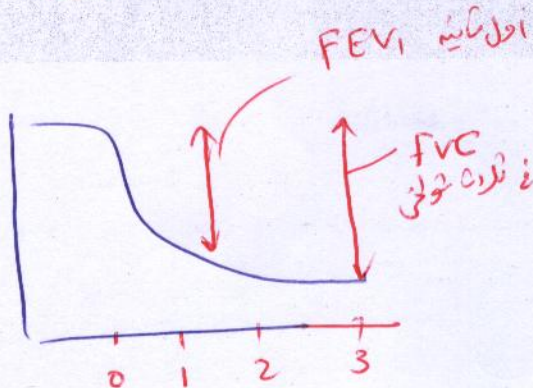
Maximum Breathing Capacity:**MBC**

- o The maximum volume of air that can be respired in one min. by the deepest & fastest breathing.
- o Normally, 120 L/min.

RESPIRATORY VOLUMES

Lung Volumes measured with a spirometer during quiet breathing with one maximum breath. Values shown are for an average-sized healthy young male.

<u>RV</u>	Residual volume	<u>FRC</u>	Functional residual capacity
<u>ERV</u>	Expiratory reserve volume	<u>TV</u>	Tidal volume
<u>IRV</u>	Inspiratory reserve volume	<u>FVC</u>	Forced vital capacity
<u>TLC</u>	Total lung capacity	<u>IC</u>	Inspiratory capacity



FVC ↓ (مقدار الهواء الخارج في أقصى تنفس)
 FEV₁ ↓ (مقدار الهواء الخارج في أول ثانية)
 FRC ↓ (مقدار الهواء المتبقي في الرئتين بعد التنفس العادي)
 FEV₁ ↓ (مقدار الهواء الخارج في أول ثانية)
 TLC ↓ (مقدار الهواء في الرئتين والقصبات عند أقصى تنفس)
 IC ↓ (مقدار الهواء الذي يمكن إضافته إلى الرئتين عند أقصى تنفس عادي)
 IRV ↓ (مقدار الهواء الذي يمكن إضافته إلى الرئتين عند أقصى تنفس عادي)
 ERV ↓ (مقدار الهواء الذي يمكن إخراجه من الرئتين عند أقصى تنفس عادي)
 RV ↓ (مقدار الهواء المتبقي في الرئتين بعد التنفس العادي)

أهم حاجتنا: صحة النفاذ

Pattern of restrictive hypoventilation in RFTs

شرح
- 5PF

- Respiratory volumes & capacities:

Decreased:  FVC $\ll 4800$ لا ينفذ
Normal:  RV. على الحزف
Decreased: TLC. $\ll 6000$

- Expiratory flow rates:

- Normal: $\downarrow$ FEV₁ / FVC. $\downarrow$

- Decreased lung compliance.

لا أدنى من لا آخذ

Pattern of obstructive hypoventilation in RFTs

شرح
- COPD
- B.A.

- Respiratory volumes & capacities:

Decreased:  FVC. $<$ تقل بمرحلة
Increased:  RV. Restri. زي
Increased: TLC. > 6000 لا ينفذ على الحزف

- Expiratory flow rates:

- Decreased: FEV₁ / FVC.
& Decrease PEF

- Increased lung compliance.

القفير

Compliance occurs against elastic recoil of lung

- COPD $\rightarrow$ $\downarrow$ elastic recoil

elastic tissue

اللي ساكن
Compliance

White Knight Love
Compliance dropped
معي حاجه ساكن

RESPIRATORY FAILURE

lab - 10-15

H.F.
n.b
essentially
clinical

1 DEFINITION

- Laboratory diagnosis characterized by:

- لازم • **HYPOXIA** (< 60 mm Hg) with or without
- عکس و متن لا • **HYPERCAPNEA** (> 50 mm Hg), (intype II) $P_{O_2} : > 85$
 $P_{CO_2} : 35-45$
- due to a disease affecting respiration.

Respir (not Resp. disease
but other disease as GBS affects

2 ETIOLOGY

اشح کاف 1- Hypoventilation.

اشح کاف 2- Impaired diffusion. E

3- Ventilation - perfusion (V / Q) mismatch.

See before

diffusion

3 TYPES

کده دلا کده لازم hypoxia

A) According to the gas defect:

1. HYPOXEMIC TYPE (TYPE I):

- **HYPOXIA:** with **normocapnia** or **hypocapnia**.

$\downarrow O_2 \pm CO_2$ ($\downarrow$ diffusion)

2. HYPERCAPNIC TYPE (TYPE II):

- **HYPOXIA:** with **HYPERCAPNIA**.
 $\uparrow$ Resp. acidosis

$\downarrow O_2 \uparrow CO_2$
 نقد، بعضی های وقت
 بهر حال زیاد
 وسایط
 $\downarrow O_2 \uparrow CO_2 \rightarrow \uparrow \uparrow RR$
 washout $\downarrow CO_2$

Most Common
as causes of it
are more common

B) According to the clinical onset:

1. ACUTE TYPE:

- Onset: acute (minutes to hours).
 • pH in Type II: markedly $\downarrow$ (respiratory acidosis).
 no time for compensation

acidosis =
 $\uparrow CO_2$
 HCO_3^-

و هذا لا
 أبدا لا
 Type II only

2. CHRONIC TYPE:

- Onset: gradual (days to weeks).
 • pH in Type II: slightly $\downarrow$ (renal compensation).

as in chronic
 time for renal compensation ($\uparrow HCO_3^-$) $\uparrow PCO_2$
 $\uparrow HCO_3^-$

Oral
 آید نوع
 R.F.
 فیه
 Acidosis
 $\downarrow$
 II only
 $\downarrow$
 آید نوع؟
 آید نوع
 Acute
 لوراکم
 و تقویم
 73

White Knight Love

4

CLINICAL PICTURE

1- Manifestations of:

HYPOXIA.

2- May be manifestations of:

HYPERCAPNEA.

3- Manifestations of the cause: **e.g.**

- Features of **COPD**: chronic respiratory failure.
- Features of **GBS**: acute respiratory failure.

double pathology → acute ascending paralysis / following infection (Campylobacter, E. coli)
+ Auto immune (Abs against myelin sheath of nerves) (glycolipids)

MANIFESTATIONS OF HYPOXIA

1. Acute hypoxia:

- BP:
- PULSE:
- RESPIRATION:
- Central Cyanosis.**
- DROWSINESS:**
- DEATH.**

Hypotension. (VC of Pulm. B.V. → VD of all peripheral B.V.)
Tachycardia, Arrhythmias → Qualitative ischemia
Tachypnea, Dyspnea.

>20

Mary's law
(↓ BP → ↑ HR)

convulsions, coma.

Persistent brain hypoxia

Central cyanosis

Blood pumped from Aorta contains

>5gm% reduced Hb

Peripheral cyanosis
normal Blood from Aorta

2. Chronic hypoxia:

- Headache. (VD stretch meninges)
- Polycythemia
- Cor pulmonale**
- Central Cyanosis.**
- Clubbing.**
- Apathy, fatigue, drowsiness.

(secondary) (by PRV)

(secondary to pulmonary hypertension).

hypoxia
↓ VC in lung B.V.
↓ P.H → RVE → RVF

hypoxia
↑↑↑ → erythropoietin

↑↑↑ BM

Polycythemia

hypoxia ↑↑↑ in i...
Pallor C...

Causes of clubbing

Chronic hypoxia
chronic toxemia (eg lung)
Br. ca.

Chronic abscess

Bronchiectasis

المرضى chronic
exacerbation



5

TREATMENT

1- Admission to hospital:

in a respiratory ICU.

2- Supportive treatment:

A) Improvement of ventilation:

1. Maintenance of patent airways. ETT
2. Elimination of retained secretions:

- 1 - Encourage the patient to cough.
- 2 - Respiratory exercises.
- 3 - Expectorants e.g. potassium iodide.
- 4 - Liquefaction of sputum:
 - Adequate hydration.
 - Mucolytics e.g. bromhexine.
- 5 - Suction of secretions through:
 - Catheters.
 - Endotracheal tube.

B) Oxygen inhalation.

C) Mechanical ventilation.

3- Treatment of the cause:

e.g.

- Treatment of COPD: chronic respiratory failure. اشبع
- Treatment of GBS: acute respiratory failure. اشبع

eg: Cortisol
eg: IV & g. globulin

4- Treatment of complications:

e.g.

- Treatment of pulmonary infections. due to COPD retained secretions.
- Treatment of acidosis: (in acute Type II RF).

by IV NaHCO₃

CHRONIC BRONCHITIS

1 DEFINITION

A condition characterized by ^① excessive mucus secretion from the bronchial tree, in which there is ^② productive cough every day, or most days, for at least 3 months of the year, for at least 2 successive years.

↓ diurnal variation
↓ winter

2 ETIOLOGY & PREDISPOSING FACTORS

(7 S)

1. **S**ex: males more than females.
2. **S**usceptibility: family history common.
3. **S**moking: (the most common cause).
4. **S**mog: atmospheric pollution.
5. **S**inusitis: chronic sinusitis predisposes to bronchitis.
6. **S**uperinfection: St. pneumoniae & H. influenza.
7. **S**ocial class: lower socio economic groups than higher.

Bronchial
irritation

8. OTHERS:

- ✓ a. Chronic lung congestion ^{↑↑ = V-V} (LVF).
- ✓ b. Chronic lung plethora ^{↑↑ = AA} (congenital heart disease with Lt to Rt shunt).
- c. Bronchiectasis.
- d. Allergic factors: may play a role.

↑↑ = V-V
↑↑ = AA
recurrent chest infects

3

PATHOLOGY

3 stages or 3 chronological types

- There are **3 types** which represent gradual increase in the severity of the disease:

1. Chronic simple bronchitis:

- There is hypertrophy of the **mucus-secreting glands**.
- The patient expectorates **whitish** mucoid sputum.

irritating
goblet

2. Chronic mucopurulent bronchitis:

- There is **secondary infection** most commonly with:
St. pneum. & H. influenzae.
- The patient expectorates **yellowish mucopurulent** sputum.

3. Chronic obstructive bronchitis:

- There is chronic **irreversible** airway obstruction, mainly affecting the **small** airways, due to:

1. Impaction of **mucus** in the lumen.
2. **Peribronchial fibrosis** & **thickening of bronchial wall**

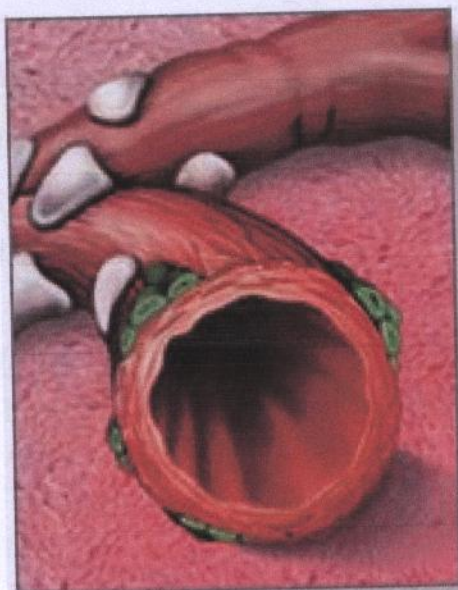
- This condition may be complicated by emphysema.

Cough
Dyspnoea
Wheezing

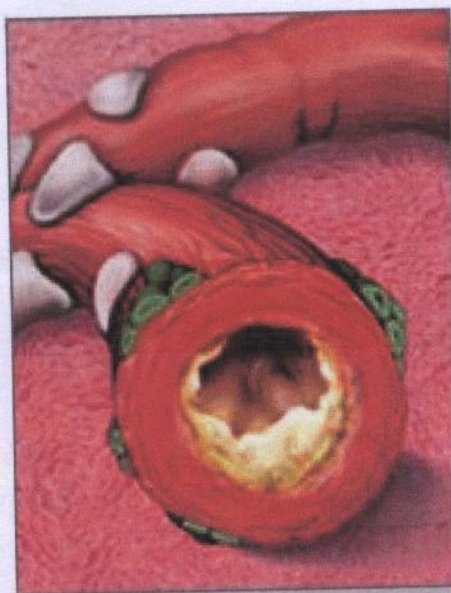
also in B.A
w/ (w occurs - attacks)

BRONCHITIS

Normal bronchi



Bronchitis



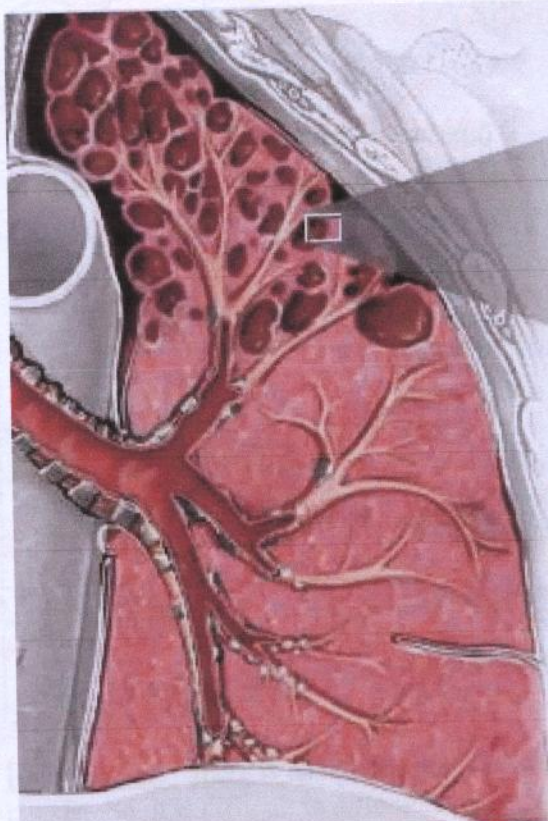
lung & liver

α₁-anti-trypsin def.

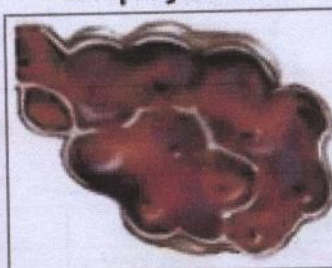
Oral

emphysema m.v. young age
liver cirrhosis " " "

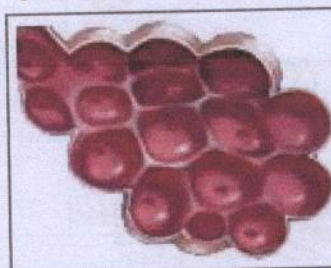
EMPHYSEMA



Alveoli with emphysema



Microscopic view of normal alveoli



lung & kidney

Good pasture syndrome

EMPHYSEMA versus CHRONIC OBSTRUCTIVE BRONCHITIS

- In emphysema:

- (only & this may occur theoretically cont Bronchitis if cause is 1st)*
- ↓ s.t. - **Impaired diffusion** due to destruction of the alveolar walls is the major abnormality leading to:

HYPOXIA & **NORMOCAPNEA**.

- Due to **HYPOXIA**, stimulation of the respiratory center occurs & results in: severe DYSPNEA which partially corrects the hypoxia.

- Conclusion:

- Presence of: severe dyspnea.
- Absence of: Central cyanosis, PH, corpulmonale, RVF. X
- This type is: "Pink puffers", Type A, or Emphysematous Type.

- In chronic obstructive bronchitis:

- (only & this occurs cont emphysema if v. early)*
- obstr. hypervent. - **Impaired ventilation** due to airway narrowing is the major abnormality leading to:

HYPOXIA & **HYPERCAPNEA**.

- Due to **UNKNOWN REASON**, stimulation of the respiratory center does not occur, this results in: absence of severe DYSPNEA.

- Conclusion:

- Absence of: severe dyspnea.
- Presence of: Central cyanosis, PH, corpulmonale, RVF.
- This type is: "Blue Bloaters", Type B, or Bronchitic Type.

due to compensatory ← *بقية بيمن كثير (أنت عنه زي)* ← *as no C.C.* ← *as polycythemia*

	Pink Puffers	Blue Bloaters
- Dyspnea	- Severe	- Very mild
- Sputum	- Small volume	- Large volume
- Central cyanosis	- Absent	- Present
- Pulmonary hypertension	- Very mild or absent	- Present
- Cor pulmonale & RVF	- Very late or absent	- Present
- Chest examination	- Hyperinflation <i>(Barrel Hyperresonant)</i>	- Wheezes <i>(vesicular & prolonged exp + wheezes)</i>
- Chest X - ray	- Hypertranslucency	- No Hypertranslucency
- ABG		
- PO₂	- Mild decrease ↓	- Severe decrease ↓
- PCO₂	- normal ±	- High ↑

diffusion surface ← *جيب هلال* ← *جيب هلال* ← *جيب هلال*

has C.C. ← *منفوخ* ← *RVF*

HOWEVER: there is usually overlap between both types because they frequently coexist. So, it is difficult to differentiate them into 2 separate clinical types.

COPD

Chronic Obstructive Pulmonary Disease COPD

سبب سريري
↓
Vcal - Asthma
↓
Diff - ILD
↓
V, D, P - COPD

DEFINITION

- A group of chronic & slowly progressive respiratory disorders characterized by:
reduced expiratory flow rates. * FEV₁
* PEFR
- It includes: chronic obstructive bronchitis, & emphysema which usually co-exist.

PATHOPHYSIOLOGY

1. Impaired ventilation:

- This is due to:

2. Impaired diffusion:

- Destruction of the alveolar walls causes:

3. Impaired perfusion:

- Destruction of the alveolar septa causes:

4. Hyperinflation:

- It is due to:
- It leads to:

increased residual volume due to obstruction.
signs of hyperinflated chest. (↑↑ Intra alv. Pn)

5. Cardiovascular abnormalities:

I. Pulmonary hypertension: due to:

1. Hypoxia: will cause:

- Direct VC of the pulmonary arteries.
- Increased blood viscosity due to polycythemia. (↑ erythropoietin)
(pressure overload)

2. Reduction of the pulmonary vascular bed: due to:

- Destruction of the alveolar septa will cause: loss of the capillaries.
- ↑ intra - alveolar pressure will cause: compression of the capillaries.

II. Hypoxic cor pulmonale. RVE is or cor failure due to lung disease

(failure) ← أدت إلى ← (Hypoxia)

2nd common cause
of RVE
is COPD

edema causes { Salt & H₂O retention < 1 Aldost. ↑
RVF 2 Kidney compensates
↑ caps permeability (hypoxia) Acidosis

6. Renal & hormonal abnormalities:

- Chronic hypoxia & hypercapnea have been shown to cause increased levels of: renin, aldosterone, noradrenaline.

- Salt & water retention will occur even in the absence of RVF.

CLINICAL PICTURE

Type of patient

- Usually a male, chronic heavy smoker, above 50 years.

Symptoms

1. Long history of chronic bronchitis:

- Chronic cough with expectoration of mucoid sputum.
- Chronic cough with expectoration of mucopurulent sputum.
- Chronic cough with gradual progressive dyspnea & wheezing.

2. Chest pain,

due to:

- Pain in intercostals muscles.. from chronic cough. strain
- Pneumothorax..... from rupture of emphysematous bullae.
- Pleurisy from complicating pneumonia.

3. Oedema of lower limbs,

due to:

- 1 • RVF.
- 2 • Salt & water retention.
- 3 • ↑ caps permeability

4. Symptoms of complications,

e.g. respiratory failure.

- hypoxia: eg Central cyanosis
- hypercapnia: eg Hypersomnia

White Knight Love

Pulsus paradoxus
Pericardial effusion
CP
P. Embolism
COPD & asthma

Pulsus paradoxus
inspiration = compliance
in COPD → ↓ elastic recoil
↑ Compliance

over expansion of lung
over expansion of pulm. V.V.
Pooling Blood
↓ Blood to heart
↓ pulse vol. in inspiration

Signs

GENERAL SIGNS

1. Vital signs:

① rhythm arrhythmia (Qualitative ischemia)

1st Commonest → A-F
2nd Commonest → irregular - absent P

↓ PR → ↓ DBP → ↑ P.P. → big pulse vol.

due to VD effect of hypoxia.

due to PH & RVF.

↓ volume → ↓ CO
↓ volume → RVF ↓ CO

Pulse:

② volume

In early cases:

- Big pulse volume:

In late cases:

- Small pulse volume:

In severe cases:

Pulsus paradoxus.

RVF لا يصل الى PH. هيقه غالياً وصل الى PH. فكمه ولا كده

Respiratory rate:

Tachypnea: with working accessory respiratory muscles.

2. Head examination:

- Puffiness of the eye lids:
- Central cyanosis:

due to chronic cough. laxity may be present.

3. Neck examination:

"Congested pulsating"

حوار Clinical

- 1 ○ Increased intrathoracic pressure. → congested only during expiration
- or 2 ○ RVF. → filling

حيث انزوم انديعج واصل الى القلب
exp & insp } filling
expiratory filling only

4. Upper limbs:

- Mild clubbing:
- Marked clubbing: 1st → 3rd degree

hypoxia is the rule.

occurs if there is associated bronchiectasis.

5. Lower limbs:

- 1 ○ RVF.
- 2 ○ Salt & water retention.

"Oedema"

hypoxia
toxemia

acquired due to both
Infects
Obstruct

Brachifera
(compensatory) COPD

White Knight Love

6. General signs of complications, e.g. respiratory failure, especially:

$\downarrow$ O_2

- **C**entral **C**yanosis.

$\uparrow$ CO_2

- **A**sterixis

(flapping tremors).

$\uparrow$ CO_2

- Hypersomnia

(CO_2 narcosis) & Drowsiness.

ABDOMINAL SIGNS

1. Liver:

- The lower border of the liver may be palpable due to:
 - a) *Ptosed liver*: pushed down by flat diaphragm due to hyperinflation (non tender).
 - b) *Enlarged liver*: due to RVF (tender). *Constitution* (other manifests of RVF)

2. Ascites:

(after edema L.L.)

- It is due to RVF.

سحب سائل
Salt & water retention

CHEST SIGNS

Inspection

- Shape:
- Movement:

Bilat Bulge
✓ barrel shaped chest.
✓ decreased bilaterally.

Palpation

- Trachea:
- TVF:

*↑ if Pneumonia
Bronchiectasis*
✓ central.
✓ decreased bilaterally all over the chest.

Percussion

- Hyperresonance:
- Encroachment on:

Bilat.
✓ all over the chest.
the normal hepatic

- U.B. 2 methods:
- ① heavy percussion (Tidal)
 - ② TVF by ulnar border of hand in spaces
- L.B. 2 methods

- ① light percussion
- ② palpation

Auscultation

- Breath sounds:
- Additional sounds: *polyphonic* generalized rhonchi, mainly expiratory, may be early inspiratory crepitations.

vesicular with prolonged expiration.

recurrent infections

normally
L 5th space in HCL.

Span
6-12 cm
4-8 cm

الهواء
↓
liver
becomes dull at 6 or 7 in

الهواء
↓
Base area of heart
↓
becomes dull resonant

الفسير
مفاتيح

- ① Smoking $\Rightarrow$ damage endothelium of bronchus (mucociliary response to clean bacteria)
- ② Retained secretion $\Rightarrow$ due to obstructive
- ③ $\downarrow$ Local immunity $\Rightarrow$ due to $\downarrow$ vasculature (rupture caps)

COMPLICATIONS

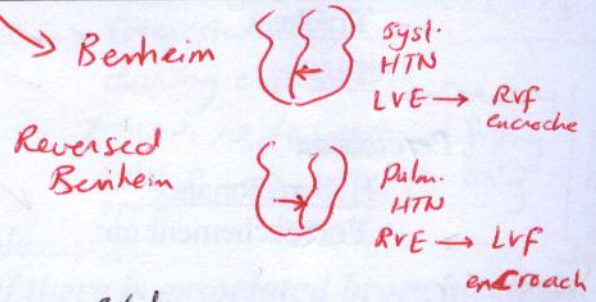
- | | | |
|---|---|------------------------------|
| 1. Respiratory failure. | } | Failure
Chronic |
| 2. Right sided heart failure following cor pulmonale. | | |
| 3. Left sided heart failure. | | |
| 4. Pulmonary infections. | } | Chest complications |
| 5. Pneumothorax. | | |
| 6. Bronchiectasis. <i>infectn obstructe</i> | | |
| 7. Polycythemia. | } | General complications |
| 8. Salt & water retention. | | |
| 9. Complications of chronic cough. <i>may and symptomatic اشرف بآلة</i> | | |

Causes of Left sided HF in COPD

1. Hypoxia: leading to myocardial ischemia. *Qualitative (weak)*
2. Acidosis: leading to myocardial depression. *(-ve inotropic $\downarrow$ contractility) catecholamines acidosis*
3. Volume overload due to: hyperdynamic circulation due to VD.
4. Increased blood viscosity due to: polycythemia.
5. Reversed Bernheim effect.
6. Associated disease of the left side of the heart, especially CAD.

الذئبة

elderly, smoking



INVESTIGATIONS

1. Chest X ray:

a) Signs of hyperinflation:

- Hypertranslucency of the lung fields. *Bilat Present IPUM*
- Transverse ribs & wide intercostal spaces.
- Low flat diaphragm.
- Heart shadow is elongated *"ribbon-shaped heart"*
- May be emphysematous bullae. *white ring jet black inside*

b) Increased bronchovascular markings.

Bronchitis $\rightarrow$ thickened wall

2. Respiratory function tests:

Specific pulmonary function tests:

a) Ventilation tests:

- Features of obstructive hypoventilation.

اشرح المربع كامل

b) Diffusion tests:

- Disturbed tests of diffusion: decreased CO transfer factor.

اشرح

c) Perfusion tests:

- Disturbed tests of perfusion.

اشح

General pulmonary function tests: (ABG)

- Hypoxia.
- Hypercapnea.
- Increased bicarbonate. *Chronic (renal Compensation)*

if acidosis → v. mild

3. Blood picture:

- May show polycythemia. ^{2ry}

*only RBCs ↑↑↑
& WBCs Platelets } normal*

*(↑↑ RBCs
WBCs
platelets)*

4. Serum alpha 1-antitrypsin:

- Decreased in primary emphysema.

5. ECG

*anhythmias < AF
Chamber enlargement: RVE, RA, LVE
associated CAD*

TREATMENT

1. Avoid further bronchial irritation:

- Stop smoking.
- Avoid atmospheric pollution.

*Strept Pn. Penicillin
H. influenzae: Augmentin
Erythromycin*

2. Treatment of complications:

- e.g. respiratory infections, antibiotics according to: C & S.
- e.g. respiratory failure. *eg: Mech. Ventilation*

اشرح كامل
لوسوال عريض

3. Symptomatic treatment:

- Expectorants, e.g. potassium iodide.
- Mucolytics, e.g. bromhexine.
- Bronchodilators, e.g. β_2 -stimulants.
- Corticosteroids, e.g. prednisone 1 mg / Kg orally daily in:
 - Patients with chronic obstructive bronchitis not responding to bronchodilators.
- OXYGEN THERAPY.

it's irreversible but we give it to relieve spasm if occurred by retained secret & infection.

- anti-inflammatory bedem

من الحبال عند
Bronchiectasis

ج- لو عند SLS
وبالعرض
COPD

Complicatn -
ج-
LVF

Respiratory failure عند الحبال دولا

ج- مقدرش أقول انه فشل

if floppy cyanosis drowsy

(lab diagnosis)

ج- من الحبال عند الحبال دولا

ج- فقط epigastric pulsation

لانه هو لو فيه اللى لو يفتح يتخطى على

اوى تقول له ادور على اى حاجه ثانية apex

ج- من الحبال دولا عند RVF؟ ولا؟ هو كبير

failure or not

if Both insp & exp filling

1. neck v.v.

2. liver

3. S₃ gallop

on +

unreliable in COPD

as COPD itself causes salt water retention & ↑ caps permeability

دقيقه

يفتح عليه الصدر

if enlarged (spongy)

(not only felt as it may be pleural)

• & Pulsating (for TR)

• Tender

→ failure ⇒ congest ⇒ stretch capsule

as pain is generalized in abdomen due to MS strain by cough.

COPD is (not reliable)

سيفوق

حوار لل clinical

ج- حالك

ج- COPD

ج-

Ch. Bronchitis + emphysema

ج- عمره

ج- elderly, smoking

atypical chronic H/o bronchitis

ج- صفت طريقه اخرى

exam: Prolonged vesicular wheezes

ج- من الحبال دولا عند الحبال دولا

exam 1. Barrel

2. HR

3. Liver ptosis (by diaphy)

حواشيفي
البرونشيتا
attacks
in between free
free
Fibrosis associated
Chronic Bronchitis

ASTHMA

أهم حقيقة
في الربو
نظرة
Status asthmaticus

DEFINITION

- It is a CLINICAL SYNDROME characterized by **recurrent episodes** of: **airway obstruction** that resolve spontaneously or by treatment.
- The clinical episodes include 3 main symptoms: **cough**, **dyspnea**, **wheezing**.
- Completely free in between attacks

PATHOLOGY

- It is a common chronic INFLAMMATORY condition of the airways.
- It has 3 main characteristics:

1. Airflow limitation:

reversible spontaneously or by treatment.

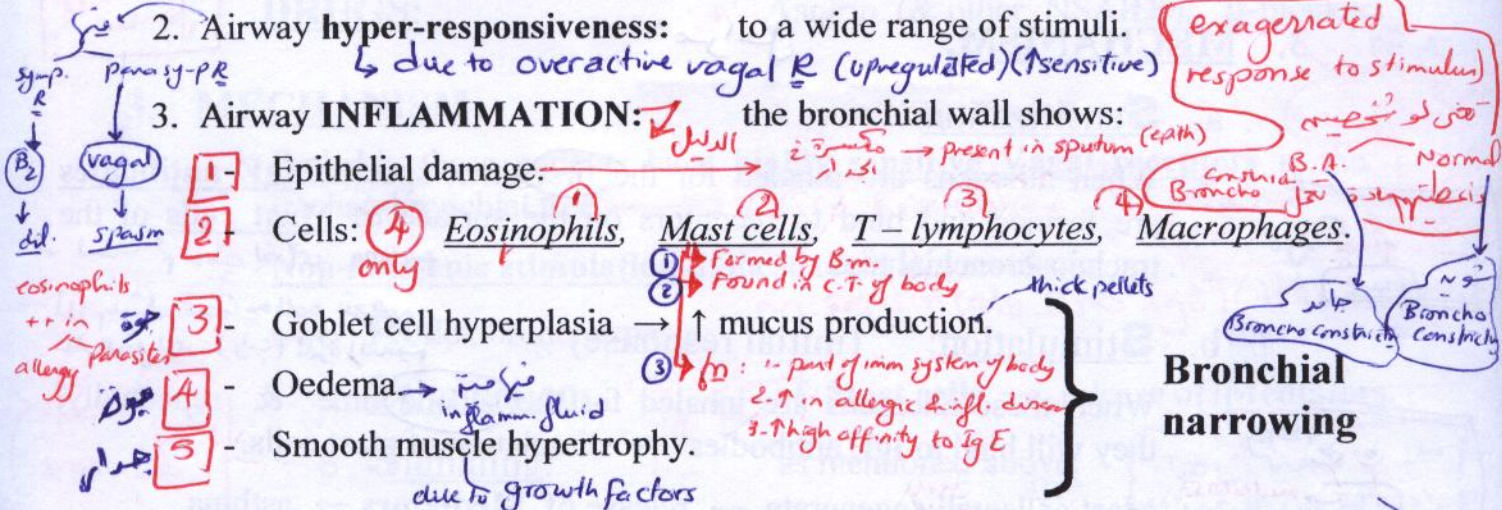
2. Airway hyper-responsiveness:

to a wide range of stimuli.

due to overactive vagal R (upregulated) (↑ sensitive)

3. Airway INFLAMMATION:

the bronchial wall shows:



With time, chronic inflammation → submucosal fibrosis → persistent airway obstruction

irreversible obstruction

CLASSIFICATION

1. Extrinsic asthma (Allergic asthma): Type I hypersensitivity

- There is a definite external cause, especially antigen or allergen.
- It occurs in ATOPIC individuals. (abnormal response in an individual of unknown etio. & predicted)

2. Intrinsic asthma (Idiosyncratic):

- There is no definite external cause, no antigen no allergen.
- Non - antigenic stimuli may be responsible.

Patients commonly show features of both types of asthma.

التهاب مزمن
inflammation
التهاب حاد
inflamed
Bronchiectasis

ETIOLOGY & PATHOGENESIS

I. Extrinsic asthma

(atopy is the major factor)

لازم حساسية
يمكن تفرغ
كلية خارجية

1. It occurs in atopic individuals:

- It runs in families.
- Associated atopic diseases: e.g. allergic rhinitis.
- Early - onset asthma: starts usually in childhood.
- Skin tests: positive.
- Serum IgE level: increased.

ليس من طرف نفس الكتل
لغات
- Rhinitis
- eczema
- asthma
only if there is allergy to certain substances

IgM: 1st response (recent)
IgG: old often 2nd exposure

2. It occurs due to exposure to allergic stimuli:

- Inhaled allergens, e.g. house dust.
- Ingested allergens, e.g. fish, eggs.
- Injected allergens, e.g. penicillin.

eg {

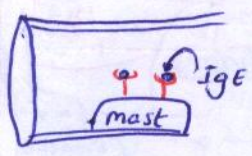
أنتج
للأنسجة
بالضيق
allergy
أنتج
الميتات
الحل للحل
أقل ال
Jumma

3. MECHANISM:

"مراحل"

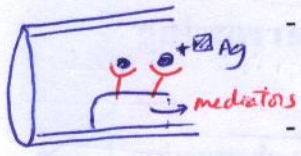
a. Sensitization:

- When allergens are inhaled for the first time, specific IgE antibodies are formed and bind to receptors on the surface of Mast cells of the tracheo-bronchial tree.



b. Stimulation: (initial response)

- When these allergens are inhaled for the second time & repeatedly, they will bind to IgE antibodies → stimulation of mast cells.
- Mast cells will degenerate → release of Mediators → asthma.



c. Signaling: (secondary response = late-onset response)

- Following stimulation of mast cells, these cells begin to signal other cells (lymphocytes, macrophages & epithelial cells) → upregulation of these cells, extension of inflammation & more release of mediators.

Cytokines released due to
4 I
infect
inflamm
injury
Immune

d. Migration: of eosinophils into the airways occurs under the effect of chemotactic cytokines.

e. Activation: of eosinophils under the effect of cytokines (IL-5)

- release of:
 - Toxic products (e.g. Major basic protein).
 - Mediators (e.g. Leukotrienes & PAF).

IL-6 in HDL in paget

f. Epithelial damage: occurs under the effect of eosinophil products.

90
White Knight Love
Bronchitis
advent
لا تفرغ كانت
سبب
no mediators

II. Intrinsic asthma

Idiosyncrasy
(airway hyper-responsiveness is the major factor)

1. It is not related to atopy or allergy:

- It does not run in families.
- No associated atopic diseases.
- Late - onset asthma: starts usually in middle age.
- Skin tests: negative.
- Serum IgE level: normal. *لو يكون على الة allergy*

2. It occurs due to exposure to non - allergic stimuli (triggers):

- Environmental: e.g. pollution & industrial materials.
- Exposure to: respiratory tract infections (*esp. viruses*).
- Exposure to: cold air.
- Exercise: **exercise - induced asthma.**
- Emotional stress: **exercise - induced asthma.**
- DRUGS: **exercise - induced asthma.**

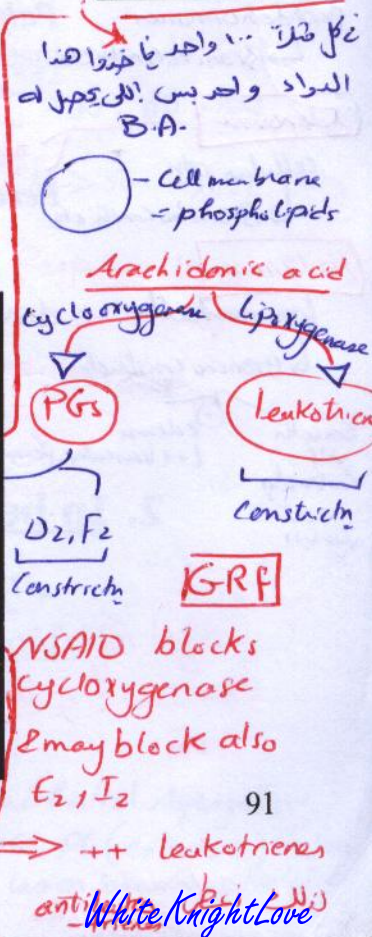
3. MECHANISM:

- Probably these patients have **highly sensitive Vagal receptors** in the tracheo-bronchial tree. *hyperresponsiveness*
- **Non-antigenic stimulation** of these receptors results in: *reflex Bronchospasm*
- **Reflex bronchospasm.** *no sensitization & no IgE (it's not atopy)*

- o **Stimulation**: of **Mast cells** → release of **Mediators**.
- o **Signaling**: as mentioned above.
- o **Migration**: as mentioned above.
- o **Activation**: as mentioned above.
- o **Epithelial damage**: as mentioned above.

Mediators involved in asthma

- Histamine.
- Adenosine.
- Serotonin.
- Prostaglandins.
- Kinins.
- LEUKOTRIENES.
- CYTOKINES (AI).
- PAF (Platelet activating factor).



تسبب
mast cells
ربو
allergic
فقط
لا
ب
هو
mast cells
تنتج
الحساسية
غير
allergy
IgE

② Airway Cooling Theory

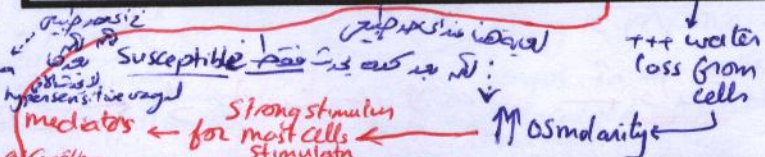
ex+++ → ++ vent.

VD rebound ← *++ cold air*

hyperemia → *edema*

Reflex Br. constriction

(++ Muscarinic R)



CLINICAL PICTURE

Symptoms

"Attacks"

- Timing:

- Usually occur in late night & early morning due to **High vagal tone**.

- Description:

Typical
Atypical

- Usually: a **Triad** of: Dyspnea, cough, wheezing.
- However: cough (esp. nocturnal) **may be the presenting feature**.
- Expectoration: viscid sputum (mucous pellets).
- Association: anxiety, sweating.

- Duration: (Variable)

- Few **minutes**..... to several **hours**or even **days**.
- Resolve spontaneously or by treatment.

Signs

1. During the attack:

"Signs of **Hyperinflation** & **Airway obstruction**"

1. Broncho constriction
2. Eosinophil attractants
3. " activated

Inspection

- **Shape:**
- **Movement:**

barrel shaped chest.
decreased bilaterally.

Kinins

peptide hormones
↳ Broncho constriction

Palpation

- **Trachea:**

central.

- **TVE:**

decreased bilaterally.

Adenosine

cellular ptn
↳ Broncho constriction

Percussion

- **Hyperresonance.**

during attack only

Histamine

from mast cells
↳ Broncho constriction

Auscultation

- **Breath sounds:**
- **Additional sounds:**

vesicular with prolonged expiration.
generalized rhonchi, mainly expiratory.

2. In between the attacks:

- The chest is usually free, **unless:**

1. Bronchial asthma is accompanied with **chronic bronchitis**.
2. Submucosal fibrosis occurs in chronic cases → persistent obstruction.

Fibrosis stage III 92

asthmatic
Bronchitis

attacks ← cough, wheezing, dyspnea

White Knight Love

Status asthmaticus

(acute severe asthma)

- It is a medical emergency:

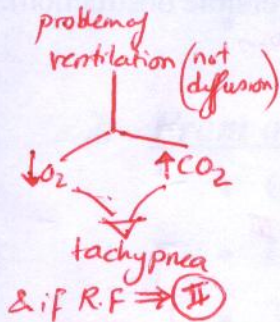
usual

The severest form of asthma that remains unresponsive to the standard treatment of asthma for several hours.

- It is of variable severity:

It may be either: severe or life-threatening.

Manifestations of severe asthma:



• RESPIRATION:

Inability to complete a sentence in one breath.

Tachypnea: RR > 25 breaths per minute. (N: 16-20)

• PULSE:

- Pulsus paradoxus.

- Tachycardia: HR > 110 beats per minute. (endogenous catecholamines)

• RESPIRATORY FUNCTIONS:

- Marked decrease of: PEFR & FEV₁ (< 50% of normal). (N: 80%)

Manifestations of life threatening asthma:

• Silent chest. = absent air entry

• Central cyanosis.

• Confusion or coma. (severe brain hypoxia)

• RESPIRATORY FUNCTIONS:

- Marked decrease of: PEFR & FEV₁ (< 30% of normal).

• ARTERIAL BLOOD GASES (ABG):

- Marked hypoxemia: PO₂ < 60 mmHg (despite ttt with O₂).

- Marked hypercapnea: PCO₂ > 45 mmHg. (N: 85-100)

* Low or falling pH.

ICU immediately
if not → death from complications

COMPLICATIONS

1. STATUS ASTHMATICUS.

2. COPD. emphysema (hyperinflated) (no bronchitis)

3. Respiratory failure. II < Acute chronic → irreversible fibrosis

4. Right sided heart failure & cor pulmonale.

5. Complications of: severe cough.

6. Complications of: therapy e.g. corticosteroids. for life

7. Pneumothorax & pneumomediastinum.

8. Allergic bronchopulmonary aspergillosis (ABPA):

③ CLP: deterioration of pt. asthmatic

④ investigate: sputum culture -ve

- CXR: localized consolidation

⑤ Ht: CBC: eosinophilia

⑥ Itraconazole serology: Anti Aspergillus Ab

① Allergic response to inhaled Aspergillus

② Fungus in an allergic pt (extrinsic) in form of localized inflammatory lesion heavily infiltrated with eosinophils

White Knight Love

INVESTIGATIONS

★ 1. Pulmonary function tests:

I. VENTILATION: Obstructive hypoventilation (Reversible):

- Decreased PEFR & FEV₁ which improve at least 15 % after inhalation of a bronchodilator (i.e. reversible airway obstruction).
- Absent response to inhaled bronchodilators occurs in:
 - a) Asthma in remission = (inbetween the attacks).
 - b) Severe longstanding chronic asthma (persistent irreversible obstruction).

II. DIFFUSION:

- CO transfer factor: normal in asthma.

III. Arterial blood gases (ABG):

- ↓ - Decreased PO₂. pH ↓ Resp. acidosis R.F. Type II
- ↑ - Increased PCO₂.

★ 2. Bronchial provocation tests:

These tests use Histamine or metacholine (inhaled) to demonstrate: airway hyper-responsiveness especially when cough is the presenting symptom.

These tests are contraindicated in severe cases with poor lung function.

3. Exercise tests:

- These tests use Treadmill to diagnose: Exercise – induced asthma.

4. Chest X-ray:

“ little role ”

- Little role in diagnosing asthma: because it is normal in between the attacks.
- During the attack: it may show signs of hyperinflation.
- May show complications: such as pneumothorax, or ABPA.

5. CBC:

- Excessive eosinophils (more marked in case of ABPA).

6. Sputum examination:

- ① Clinically thick pellets
- ② - Excessive eosinophils.
- Curshman's spirals.
- Charcot – Leyden crystals.
- Criola bodies.

7. Skin tests:

- To identify the allergen in extrinsic asthma.

8. Serum IgE:

- Increased in most cases of extrinsic asthma.

① normal
to compare e results after test (< or > 20%)
② لا سعة الكالات severe
③ خلايا
as eosinophilic in cardio variant angina

DIFFERENTIAL DIAGNOSIS

1. From other causes of paroxysmal dyspnea with wheezing:

- Cardiac asthma. *PND (2nd stage) + اشج*
- Uremic asthma.
- ~~Recurrent pulmonary emboli.~~
- Carcinoid syndrome.
- Pulmonary vasculitis.

2. From other causes of paroxysmal dyspnea: *cont wheezing*

- Cardiac dyspnea. *classic PND (1st stage)*
- Extrinsic allergic alveolitis. (farmer's lung) → *اشج → attacks then fibrosis*
- Tetany (laryngismus stridulus). *progressive dyspnea*
- Myasthenic crisis.
- Recurrent pulm. emboli. → *(only dyspnea & wheezing) or, smokes, old & typical H/O of Ch. Bronchitis*

Chronic & causes to acute

3. From chronic bronchitis.

1 Cough, dyspnea, wheezes but persistent

4. Differentiate between: *اشج / خفس سطور*

extrinsic

& intrinsic asthma.

TREATMENT

- General
- Drugs
- Long term ttt
- ttt status asthmaticus

Causes of tetany only 3

- 1 hypercalcaemia
- 2 hypomagnesaemia
- 3 alkalosis

1 GENERAL MEASURES

- Avoid exposure to stimuli: whether allergic or non-allergic.

- Treatment of: CHEST INFECTIONS.

- PSYCHOTHERAPY.

- IMMUNOTHERAPY:

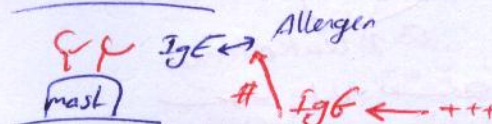
= (de) sensitisation
(HYPOSENSITIZATION)

- Repeated injection of extracts of the allergen in gradually increasing doses may be beneficial..... why ??

- It results in production of IgG antibodies which will prevent binding of the allergen to IgE (blocking antibodies).

*اشج
allergen
by skin test*

Blocks



2 DRUGS

1. BRONCHODILATORS

A) Sympathomimetics:

1. Selective β_2 -stimulants: [INHALED]

THEY HAVE A RAPID ONSET OF ACTION & FEW SIDE EFFECTS. THEY COULD BE GIVEN PARENTRALLY IN STATUS ASTHMATICUS.

- Salbutamol: (puff = 100 μ g, 2 puffs tds). } 3/d
- Terbutaline: (puff = 250 μ g, 2 puffs tds).
- Salmeterol: (puff = 50 μ g, 1 puff bid) LONG ACTING. } 2/d

2. Non Selective β_2 -stimulants:

THEY ARE OF LITTLE USE BECAUSE OF THEIR SIDE EFFECTS.

- Adrenaline: 0.5 c.c. 1 / 1000 solution SC.
- Isoprenaline: 10 mg SL or by inhalation.
- Ephedrine: 30 mg orally.

→ Catechols
↑ BP
↑ HR
anhythmias

B) Xanthines:

(Theophylline or Aminophylline)

- Short acting: slow IV infusion in status asthmaticus.
- long acting: orally for long - term control.

direct smooth muscle Relaxant

2. RECEPTOR ANTAGONISTS

A) Anticholinergic bronchodilators: "Acetylcholine antagonists"

- Ipratropium bromide inhaler (20 - 40 μ g tds).
- Oxitropium bromide inhaler.

B) Antihistamines:

"H₁ receptor antagonists"

- Terfenadine (rarely used).

C) Antileukotrienes:

- Montelukast (Leukotriene receptor antagonist).
- Zileuton (Leukotriene synthesis inhibitor).

ACE I & ARB_s

Monotherapy in mild persistent asthma, Add - on therapy in moderate to severe asthma.
Useful in: aspirin - induced asthma.

↑ Leukotrienes
2 pathways

for potentiation (synergism)

White Knight Love

3. ANTI - INFLAMMATORY DRUGS

A) Corticosteroids:

Anti fibrotic
Anti edematous
Anti allergic

1. Inhaled corticosteroids:

DRUG:

- or
- Beclomethasone (puff = 50 µg, 2 puffs tds).
 - Fluticasone.

SIDE EFFECTS:

- Hoarseness of voice. (irritates vocal cord)
- Oropharyngeal candidiasis. (↓ local imm. of Resp. tract)

2. Oral corticosteroids:

DRUG:

- Prednisone: 1 mg Kg / day.

SIDE EFFECTS:

- Refer to: Rheumatoid arthritis.

3. Parental corticosteroids: (for Status asthmaticus)

DRUG:

- Methylprednisolone. *1 mg*
- Hydrocortisone.

SIDE EFFECTS:

- Refer to: Rheumatoid arthritis.

Side effect: Cushing

B) Other anti-inflammatory drugs:

- Gold, Methotrexate, Cyclosporine.
- Under trial for severe chronic asthma not responding to other drugs.

*ILD Cyclophosphamide
TB cycloserine*

*used as imm: Behcet
Organ Transplantation
syndrome*

*side effect: nephrotoxic: GN
used in: R.A.*

*main side effect: hepatotoxic
used in: RA*

*megakaryoblastic anemia
(F.A. antagonist)
White Knight Love*

4. PROPHYLACTIC DRUGS

A) Disodium cromoglycate:

- It inhibits release of mediators from mast cells (mast cell stabilizer).
- Dose: inhaled puff = 1 mg, 2 puffs / 6 hours.

B) Kitotifen:

- It inhibits release of mediators from mast cells (mast cell stabilizer).
- It has antihistaminic effect.
- Dose: oral tablet = 1 mg, 1 tablet bid.

C) Omalizumab:

(Recombinant Anti – IgE)

- It combines with free IgE blocking its interaction with mast cells.
- Dose: single injection, (SC every 2 – 4 weeks).

3 LONG TERM CONTROL (ttt of chronic asthma)

- Inhaled β_2 – stimulants (long acting) / Inhaled Disodium cromoglycate.

IF THERE IS NO RESPONSE: ADD:

- Inhaled corticosteroids.

IF THERE IS NO RESPONSE: ADD:

- Oral theophylline (long acting).

IF THERE IS NO RESPONSE: ADD:

- Oral corticosteroids OR Inhaled anticholinergic drugs.

Antileukotrienes: helpful in any step to reduce the asthmatic symptoms.

Synergistic

but not alone

لا ينفذ بمفرده

Acute attack: responds to #1
 #1: ① inhaled β_2 stimulants + IV Aminophylline
 if no response
 ② inhaled corticosteroids

99

4 TTT OF STATUS ASTHMATICUS

if no Response
 Status asthmaticus
 وهو حالة
 حادة

A) HOSPITALIZATION: in a respiratory ICU.

B) DRUGS:

1. β_2 - stimulants:

o **IPPB:** using Nebulizer, 5 mg with O_2 .

OR

o **Parental:** IV infusion, 250 ug over 10 minutes,
 (If no improvement occurs with Nebulizer)

Salmeterol - short acting

"SALBUTAMOL"

SQ → Def + IgE + (allergic)
 LQ → Def + types + (allergic)
 ⊕ after control of S.A. → treat Pt. for asthma
 long term controller

استخدم اول مع بعد لولا نتيجة
 Add Cortisol
 ↓
 if no
 Mech. vent.

2. Inhaled anticholinergics: "IPRATROPIUM BROMIDE"

o **IPPB:** using Nebulizer, 250 ug with nebulized salbutamol.

Intermittent positive pressure Breathing turns liquid medicatns → mist breathed airway

3. Aminophylline: (Short acting, continuous IV infusion, rarely used)

o Loading dose: 5 mg / kg over 30 minutes.

o Maintenance dose: 0.5 mg / kg / hour.

Receptor antagonist

v. Slowly
 A
 B-stimulat
 ca
 Digitali

IF THERE IS NO RESPONSE: ADD:

4. Systemic corticosteroids:

o **Methylprednisolone:** 2 mg / Kg IV followed by 1 mg / Kg / 6 hours.

Reliever

o **Hydrocortisone:** 200 mg IV & repeated / 6 hours.

Controller

o **Oral prednisone:** AFTER IMPROVEMENT

1mg / Kg / day orally is given for 2 weeks, then the dose is gradually tapered by 5 mg every 3 days.
 if 60kg → 60 mg (الجرعة) = 12 غرض → كل غرض في 3 أيام

لأنه يجب أن يكون هناك تحسن تدريجي
 gradual reduction
 أما لو أفلح مع 2 أسابيع
 gradual reduction

C) SUPPORTIVE MEASURES:

1. Oxygen inhalation.

2. Correction of:

Resp. acidosis

electrolyte imbalance.

والتي تحتاجها في انت تعالج
 IV NaHCO₃

hypokalemia
 as: ① salbutamol (↓ K)
 ② Cortisol (↓ K)
 (aldosterone like on kidney)
 راقب الـ K في 6 ساعات

D) MECHANICAL VENTILATION:

- May be needed in severe resistant cases.

لشفو

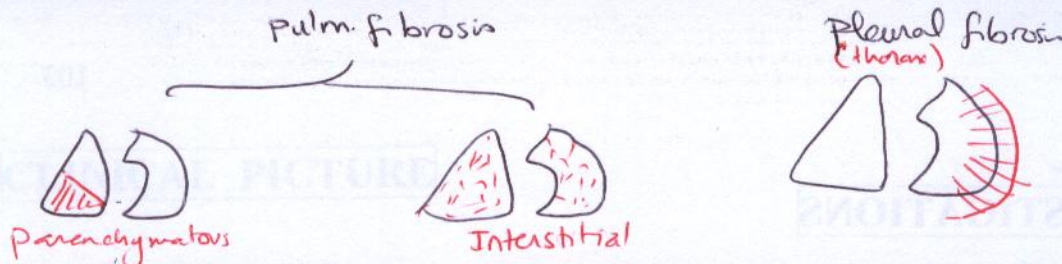
CLINICAL CLASSIFICATION OF DRUGS

1. RELIEVERS: used to relieve the **ATTACKS**

- e.g. Short acting Inhaled β_2 stimulants,
Inhaled anticholinergics,
Short acting xanthines,
IV corticosteroids.

2. CONTROLLERS: used for **MAINTENANCE**

- e.g. Long acting Inhaled β_2 stimulants, Disodium cromoglycate,
Inhaled & oral corticosteroids,
Long acting xanthines,
Antileukotrienes.



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PULMONARY FIBROSIS

Clinical
TB, الجوار، صيفر على
ILD ← نجرى

ETIOLOGY

1 Pulmonary fibrosis

I. Parenchymatous fibrosis:

- Occurs as a complication of various lung diseases, e.g. TB, bronchiectasis, lung abscess.

II. Interstitial fibrosis (ILD):

- Occurs in between the alveoli, in the interstitial tissue.

2 Pleuro-Pulmonary fibrosis " fibrothorax "

- Pleural diseases may heal by extensive fibrosis that extends to the lung parenchyma, usually unilateral.
- It commonly complicates hemothorax & empyema. & TB

Parenchymatous fibrosis & Fibrothorax

CLINICAL PICTURE

Symptoms

- Dyspnea ↓ compliance
 - Dry cough cough R pleura (Fibrothorax)
 - Chest pain (in fibrothorax). " " Paragochia (Pulm.)
- as only pleura has pain R.

Signs

Inspection:

- Shape: ✓ retraction on the affected side.
- Movements: ✓ diminished on the affected side.

Palpation:

- Trachea: ✓ shifted to the same side.
- TVF: ✓ diminished.

Percussion:

- ✓ Heterogenous dullness. patchy → (DD from collapse)

Auscultation:

- ✓ Breath sounds: decreased.
- ✓ Additional sounds: crepitations.

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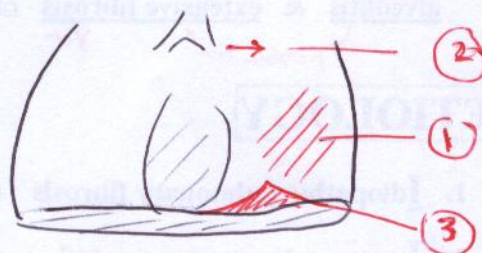
COMPLICATIONS

1. Bronchiectasis. → fibrosis traction on airways
2. Cor pulmonale. → ↓ compliance → hypoxia → VC PH RVE RVF
3. ~~Contraction collapse.~~

INVESTIGATIONS

1. Chest x ray:

- Heterogenous opacity with reticulations.
- The mediastinum moves towards the fibrosis (shift of trachea to the side of fibrosis).
- The diaphragm moves towards the fibrosis (tenting of the diaphragm).



2. Respiratory function tests:

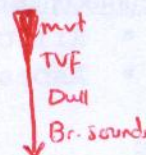
- Evidence of restrictive hypoventilation.



DIFFERENTIAL DIAGNOSIS

- From other causes of "syndrome of multiple negatives":

Lung fibrosis, lung collapse & pleural effusion.



TREATMENT

- Treatment of the cause.

Interstitial Lung Fibrosis (ILD)

DEFINITION

- Bilat Diffuse inflammatory disorders of the lower respiratory tract characterized by alveolitis & extensive fibrosis of the interstitial tissue.

Parenchymatous

- localized

→ All alveoli
interst. Branch

ETIOLOGY

Hamman Rich Syndrome

- Idiopathic pulmonary fibrosis (IPF).** (Autoimmune: responds to steroids / Immunosuppressants)
- Immune diseases:**
 - Collagen diseases. *esp SLE, scleroderma* → most common collagen disease (Abs → Alveolitis)
 - Good Pasture's syndrome. *Abs → B.m. → GN*
- Inflammatory bowel diseases:**
 - Crohn's disease. *all GIT*
 - Ulcerative colitis. *only colon*

→ Have Intest. & extraint. manifests.
- Inhalational lung diseases:** *Chronic exposure*
 - Extrinsic allergic alveolitis** (*due to organic dust*):
 - Farmer's lung. *أبيض الرئة الحبيبي الحاد*
 - Pneumoconiosis** (*due to inorganic dust*):
 - Silicosis. *زفر*
 - Asbestosis. *سراطة*
- Iatrogenic:**
 - Amiodarone.
- Hepatic diseases:** (*Autoimmune*)
 - Chronic active hepatitis. *Anti Liver kidney Abs*
 - Primary biliary cirrhosis. *Chronic autoimmune ⇒ Lupoid*
Abs against liver cells ⇒ associated autoimmune disease
anti mitochondrial Abs
- Heart diseases:**
 - Chronic left ventricular failure. *MS defensive mechanisms*
- Sarcoidosis.** *non caseating granuloma* → *Bilat enlarged Mediastinal LNs*

Sarcoidosis

- RFever
- Erythema nodosum
- DCH, RCH

RESPIRATORY FUNCTION ABNORMALITIES

- IMPAIRED DIFFUSION (*Hypoxia with normocapnea*). *Type I*
- Restrictive hypoventilation.

alv. cap membrane

Lung Restriction

لا يوجد نقص في
diffusion

106

II مع نقص
ventilatory

White Knight Love

CLINICAL PICTURE

Symptoms

- Dyspnea (gradual & progressive).
- Dry cough. $\rightarrow$ alveolitis

Signs

- Central cyanosis. $\rightarrow$ hypoxia
- Clubbing. $\rightarrow$ hypoxia
- Crepitations (diffuse). $\rightarrow$ alveol
در هر نَفَس
رطوبتی
لا فیبروز
هو

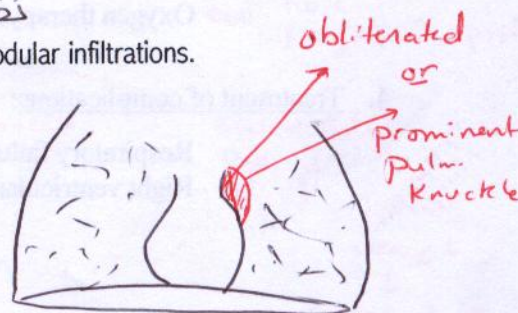
COMPLICATIONS

1. Respiratory failure. $\rightarrow$ except if v. severe hypoxia $\rightarrow$ II
2. Right ventricular failure (cor pulmonale). $\rightarrow$ Severe hypoxia $\rightarrow$ VC $\rightarrow$ PH $\rightarrow$ RVE $\rightarrow$ RVF

INVESTIGATIONS

1. Chest x ray:

- Bilateral diffuse lung affection in the form of reticulations & micronodular infiltrations. (ground-glass appearance).
- Pulmonary hypertension $\pm$ right ventricular enlargement.

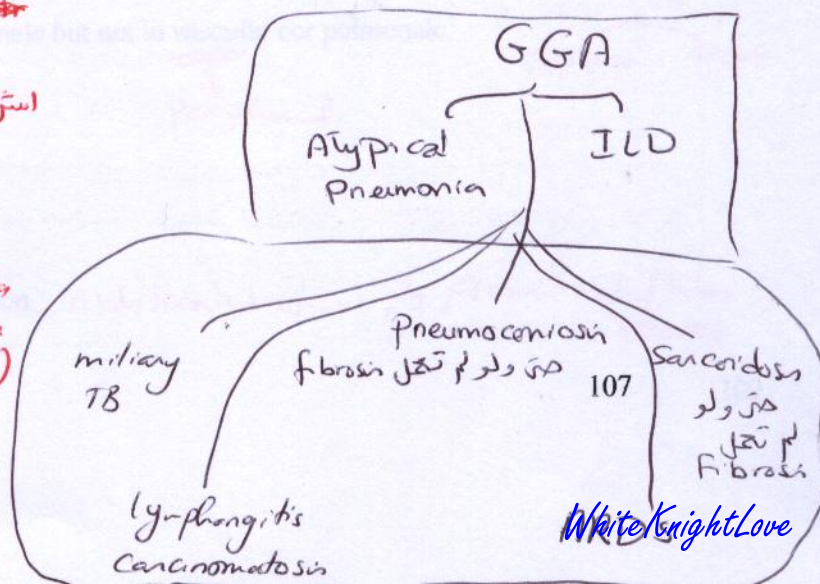


2. Pulmonary function tests:

- Ventilation tests:
Restrictive hypoventilation. $\rightarrow$ اشع
المرح
- Diffusion tests:
Decreased CO transfer factor. $\rightarrow$ اشع

3. Arterial blood gases:

- Hypoxia.
- Normocapnea. (or Hypercapnia if severe hypovent.)



4. Assessment of disease activity: (active alveolitis)

also in Pneumocystis Carinii
Angina
Technique, Mallin
P. F. bulbs
Xenon, Albumin

- BAL: for detection of type & amount of inflammatory cells in active alveolitis.

- Gallium-67 scan:

- o The affected lung tissue (with active alveolitis) takes up Gallium-67.
- o The normal lung tissue does not take up Gallium-67.

- Open lung biopsy: for detection of active alveolitis.

neutrophils
lymphocytes
macrophages
plasma cells
fibroblasts

5. Investigations for the cause.

eg: Good Pasture syndrome (Abs against Glomerular B.m.)
eg: Scleroderma (Anti SCL 70)

TREATMENT

1. Treatment of the cause.

eg: scleroderma (corticosteroids)
→ if active disease: steroid

2. Treatment of Idiopathic Pulmonary Fibrosis:

- o Corticosteroids: prednisone: 1 mg / kg / day.
- o Cytotoxic drugs: e.g. cyclophosphamide.

anti infl.
anti fibrotic

3. Symptomatic treatment:

- o Oxygen therapy.

steroid

Drug
steroid

4. Treatment of complications:

- o Respiratory failure. eg: mechanical ventilator
- o Right ventricular failure.

eg Digitalis
Diuretics

استرج
لوجوال كبر

COR PULMONALE

Clinical

DEFINITION

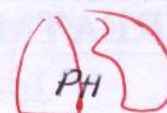
Right ventricular hypertrophy with or without failure resulting from pulmonary hypertension due to parenchymal or vascular lung disease.

ETIOLOGY

A) Acute cor pulmonale:

1. Massive pulmonary embolism.
2. Tension pneumothorax.
3. Acute massive lung collapse.

no Acute massive MI as it's not lung disease



B) Subacute cor pulmonale:

1. Lymphangitis carcinomatosa.
2. Recurrent showers of pulmonary emboli.

(Blood spread of malignant deposits to small caps of lung & peripheral lymphatics also)



C) Chronic cor pulmonale:

1. Hypoxic cor pulmonale:

Occurs in almost all cases of chronic lung diseases, e.g.

- Obstructive hypoventilation e.g. COPD.
- Restrictive hypoventilation e.g. ILD.

2. Vascular cor pulmonale:

- Primary pulmonary hypertension. (2ry more common)
- Pulmonary bilharziasis.
- Pulmonary vasculitis. → EAO → PH
- Sickle cell anemia. → sickle RBCs
- Recurrent showers of pulmonary emboli. → subacute & chronic

→ C.C.

no C.C. if cyanosis → peripheral

CLINICAL PICTURE

- 1- Features of the cause. e.g. COPD, asthma.
- 2- Features of pulmonary hypertension. → ↑ pressure
- 3- Features of right ventricular enlargement.
- 4- May be features of right ventricular failure.
- 5- Cyanosis is present in hypoxic cor pulmonale but not in vascular cor pulmonale.

سعال
neezing
↓
occlude B.v.
esp
Spleen Bone

INVESTIGATIONS

1. Chest X-ray:

- RA enlargement.
- RV enlargement.
- Features of the pulmonary hypertension.
- Features of the cause.

obliterated waist, ± Prominent Pulm Knuckle

Peripheral

2- ECG:

- RA enlargement (p- pulmonale).
- RV enlargement.

tall peaked p-wave

3- Echocardiography:

- RA enlargement.
- RV enlargement.

TREATMENT

- 1- Treatment of the cause.
- 2- Treatment of pulmonary hypertension:
 1. Vasodilators: e.g. calcium channel blockers.
 2. Anticoagulants: in cases of recurrent pulmonary emboli.
 3. Heart-lung transplantation: in advanced cases.
- 3- Treatment of right-sided heart failure. *eg Digitalis & Diuretics*

HYPOXIC VERSUS VASCULAR COR PULMONALE

Clinical

حوارف

	Hypoxic cor pulmonale	Vascular cor pulmonale
Dyspnea	Marked <i>اعياء COPD</i>	Minimal or absent
Sputum	Mucopurulent <i>سعال وديم</i>	Absent
Cyanosis	Central due to hypoxia	Peripheral due to low CO <i>متدلو</i>
Clubbing	Common	Absent <i>لوفين</i>
Chest Examination		
• Shape	Barrel-shaped	Normal
• Breath sounds	Prolonged expiration	Normal
• Additional sounds	Wheezes	Absent
• Features of PH	May be masked <i>chest hyperinflation</i> <i>لو حاجه تنفس</i>	Marked <i>متدلو</i>
Pulmonary functions	Hypoxia, may be hypercapnia	Usually normal

Bilharzial Cor Pulmonale

ETIOLOGY

Both

mansonii اکثر من hematobium
hematobium من المناعة i mansonii من
(toxins M)

1. Schistosoma mansoni ova:

- They reach the lungs only in the presence of portal hypertension & porto-systemic collaterals which allow the ova to bypass the liver to the lungs.
- They cause more destructive lesions than *S. haematobium*.

int. vv. → portal vv. →

من الدم
systemic
vv.

2. Schistosoma haematobium ova:

- They reach the lungs more commonly since the worms live in pelvic venous plexuses which drain into the lungs.
- They usually produce mild lesions.

الا لو
منه
shunt
منه تروح
long

PATHOLOGY

1. Pulmonary hypertension:

due to:



Toxins

- The bilharzial ova reach the lungs as emboli leading to necrotising arteriolitis due to the secretion of miracidial toxins. This is followed by end-arteritis obliterans with obliteration of pulmonary arterioles leading to pulmonary hypertension.

Complex

- Bilharzial immune complexes may play a role through causing pulmonary vasculitis, leading to pulmonary hypertension.

→ Ag + Ab + Complement

A. 2
مخبره

2. Aneurysm formation:

- Aneurysmal dilatation of the pulmonary artery & its two main branches occur due to progressive weakness in the wall of the arteries.

due to toxins & complexes } → A. كبير
ليس لانه A. كبير
لا يقفل كما يوسع

A. 3
كبير

3. Cor pulmonale:

- Pulmonary hypertension may lead to right ventricular enlargement & eventually failure (cor pulmonale).

CLINICAL PICTURE

Symptoms

- Symptoms of low CO. *Even CO failure* 2-1
- Symptoms of right ventricular failure may occur late! 2-1
- No central cyanosis. *(may be peripheral)*

Signs

- Signs of pulmonary hypertension (refer to cardiology).
- Signs of pulmonary aneurysm: *marked pulsations & dullness in the left second left space.*
- Signs of right ventricular enlargement.
- Signs of right ventricular failure.
- *Cause* → Hepatosplenomegaly & signs of portal hypertension. *varices & hematemesis*

INVESTIGATIONS

1) Chest x-ray:

- Marked dilatation of the main pulmonary arteries forming: *aneurysmal*
"Dumb-bell shaped shadows in the hila".



D.D
hilar
Bilat.
LNS
enlargement

- *Also in pericardial effusion* → Peripheral pulmonary oligoemia.
- RV enlargement.
- RA enlargement.

↓ IPVM due to obliteration of pulm. Arterioles

2) ECG:

- RV enlargement.
- RA enlargement (p – pulmonale).

3) Investigations for bilharziasis:

(refer to GIT).

L antischistosomal Ab_i (Serology)

TREATMENT

- 1) Proper treatment of cor pulmonale.
- 2) Anti-bilharzial treatment.

*اشترج
كابل*

Pneumonia
 Br. ca
 B.A.
 TB

واحد من أهم الأمراض

113

TUBERCULOSIS

1

DEFINITION

- It is a **common** ^{infectious} contagious infection caused by **MYCOBACTERIUM TB**.
- It primarily affects the **LUNGS**, however: infection can spread to other organs.

نخرى مش كابل
 من حيث من
 أي حنة
 تشقوى
 (Anti-TB drug & # status Asthmatic)

2

ETIOLOGY

1. Causative organism:

MYCOBACTERIUM TB

- **Acid** - fast.
- **Alcohol** - fast.
- **Aerobic**.

organism after staining is not decolorized by Acid or Alcohol

so it's apical in lung (as O_2 Tension is $\uparrow\uparrow$ in apex)

(also Klebsiella)

2. Source of infection:

- Patients with **Active pulmonary TB**, especially with **cavitary lesion**.

Aggressive

if latent no infect

if fibrosis no infect or it

3. Mode of infection:

- **Airway Droplet** infection, especially with **prolonged contact** (weeks).

3

PREDISPOSING FACTORS

1. Age:

- Below 5 y:
- 5 - 15 y:
- Above 15 y:

High susceptibility to infection

Less susceptibility (golden age) as $\uparrow$ imm

High susceptibility to progressive pulmonary disease

يكتب كاملاً مع سؤال etio

لسوا
 دخل
 ربي
 وده
 لا يدخل
 active

2. Race:

more common in **black races**.

3. Incidence:

"Poor countries & Poor socio-economic classes"

Poor nutrition,

Poor hygienic conditions.

↓ nutrition
overcrowded

overcrowded

غلب
 بيخرب
 active

113

TB اعتبره
 until proved otherwise
 negro
 or DM

White Knight Love

4. Occupation:

- **Paramedical & medical** people are more exposed to infection.

لازم تاخذ vaccine
اعلى tuberculosis

5. Chest diseases:

- **Pneumoconiosis:** e.g. **Silicosis, asbestosis** ↓ resistance to infection.
- **Pulmonary oligemia:** e.g. **PS.**

تؤثر cells التي تدافع عنك

↓ alveolar macrophages
& lymphocytes
due to ↓ vascularity of lung
(Fibrosis)

6. Decreased immunity:

- **Diabetes mellitus.** (TB is shadow of DM)
[طلا وجد DM تراحد TB]
- **Drugs:** Corticosteroids & other immunosuppressives.
- **AIDS.**
- **Malignancies,** especially lymphomas & leukemia
- **Malnutrition.**

PATHOGENESIS

I. ENTRY

Inhaled TB bacilli travel via the airways to be deposited in the pulmonary parenchyma, especially in the **upper zones**, probably due to high O₂ tension which favours the growth of the organisms.

as obligate aerobe
يعتبر او
anaerobic metabolism

II. FIGHT

Alveolar macrophages ingest TB bacilli & try to phagocytize them.

Alveolar macrophages usually fail to completely destroy the bacilli which possess natural defences. They are usually **not destroyed.**

There is a fight between the: **Alveolar macrophages & TB bacilli.**

only macrophages
inactivate TB bacilli
not kill it

دلو ليس تاني reactivate

III. FATE

1. Primary TB:

(Childhood) < 5y

a) Latent form:

In most immunocompetent individuals, macrophages are successful in inactivating the bacilli. The bacilli stay LATENT or DORMANT. Therefore the infection is self-limited and often subclinical. Patients are asymptomatic and the underlying lung pathology is: The primary complex: Gohn's focus, regional lymphadenitis, lymphangitis

b) Active (Progressive) form:

In some patients, macrophages fail to inactivate the bacilli. The bacilli become active leading to a clinically apparent infection.

Patients may present with: pulmonary (or) extrapulmonary manifestations

2. Postprimary (reactivation) TB:

(Adulthood) > 15y

After a period of latent primary TB, reactivation of the disease may occur at any time if the immunity of the patient drops.

Patients may present with: pulmonary (or) extrapulmonary manifestations

PATHOLOGY

1. Primary TB: (Latent) (if active) (Childhood)

a) Gohn's focus: (pulmonary component)

An area of consolidation formed by aggregation of tubercles, present usually in the mid to upper zones, usually subpleural.

b) Regional lymphadenitis (glandular component)

c) Lymphangitis in the intervening lymphatic vessels.

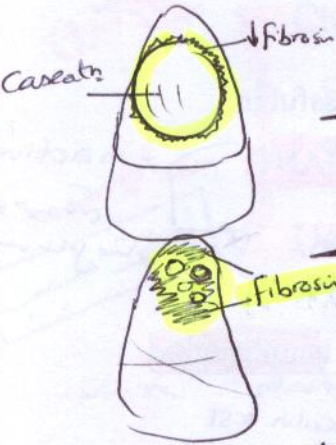


الجزء
complex
الى اسم
هو دة
lung

= macrophages
engulfing
TB bacilli

2. Postprimary (reactivation) TB: (Adulthood)

a) Gross: (autopsy)



- This depends on the state of immunity & the virulence of the organism:

1. FIBROCASEOUS TB: (↓ bad immunity) (Apical cavity)
 - Big **cavity** surrounded by **little** fibrosis.
2. FIBROID TB: (↑ good immunity) (Apical fibrosis)
 - Small **cavities** surrounded by **excessive** **fibrosis**.
3. Endobronchial TB:
 - TB bronchitis of the bronchi connected to TB cavities.
4. TB bronchopneumonia & Miliary TB:
 - These types occur in **immunocompromised** patients.

b) Microscopic:

- The characteristic lesion is: "Typical TB granuloma"

- CASEATION: central, surrounded by:
- CELLS: ^{inflammatory} Lymphocytes, Langerhan's giant cells, Fibroblasts.
- CALCIFICATION.



CLINICAL TYPES

1. Primary TB:

(Childhood)

a) Latent form:

Patients are usually **asymptomatic**.

b) Active (Progressive) form:

Patients may present with: pulmonary or extrapulmonary manifestations

2. Postprimary (reactivation) TB:

(Adulthood)

a) PULMONARY TB.

b) EXTRA - PULMONARY TB.

Signs

1. General signs:

• Fever,

• Clubbing:

• PALLOR. → anemia

as lung abscess & Bronchiectasis

؟ pale (سفيد)
miliary TB : ٥١ -
Cyanotic

Pallor in chest
↳ malignancy
↳ infects → pneumonia abscess Bronchiectasis

GRF anemia in

1. Anemia of chronic disease (ACD)

2. Toxic inhibition of TB (Aplastic)

3. Anorexia

4. Extra pulmonary spread (infiltrating B.m.) [myelophthisic]

5. hemoptysis [reddefecating anemia]

toxic facies, loss of weight.

in advanced untreated cases with severe toxemia.

لذا کم کریں
vit B12
Folic Acid

anorexia
toxemia
catabolism

2. Chest signs:

(variable depending on the lesion)

• Apical:

cavitation,

fibrosis.

(the most common)

• Others:

consolidation,

bronchopneumonia,

bronchiectasis,

pleural effusion,

pneumothorax.

Bilat patchy opacity

parenchymatous
not ILD

انسج لانسی
کامل

COMPLICATIONS

1. AMYLOIDOSIS

(Secondary).

Extra pulm → renal amyloidosis
Toxemia (even if restricted TB to lung)
↓
renal amyloidosis

2. Aspergilloma

(formed in a TB cavity).

imm → fibrocarcous means that imm is
fungi

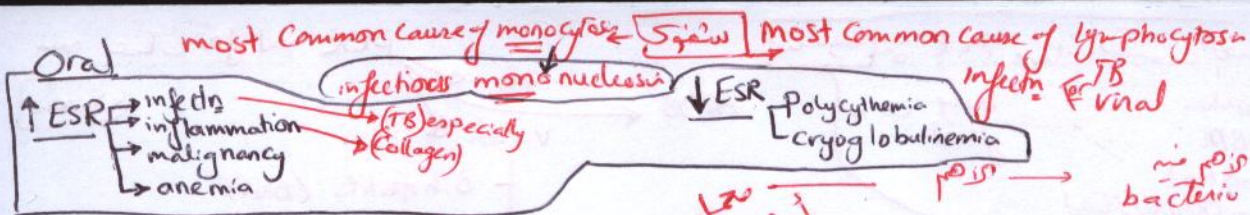
فستق ای
فنج: mild
Fungal clumps
" Ball
: ilia - conazole

3. SPREAD

(Extra - pulmonary TB).

4. COMPLICATIONS OF ANTI - TB drugs.

- کُشع کامل لوسوال درجه کبیر
- کُشع فقط : eg لوسوال ۵ درجه



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INVESTIGATIONS

I. LABORATORY INVESTIGATIONS

1. General:

(evidence of tissue inflammation)

a) ESR: markedly elevated.

b) CRP: elevated.

c) CBC:

- WBCs: lymphocytosis & monocytosis.
- RBCs: anemia.

Acute phase Reactants

lymphocytes & monocytes

immune response to TB (cell mediated)

also ↑ in RF

2. Bacteriology:

a) Sputum: for TB bacilli

- Staining: ZN stain.
- Culture: Lowenstein-Jensen medium or BACTEC.
- Animal Inoculation: Guinea pig.

if +ve: ok
-ve: not considered -ve except if 3-6 times repeated (do not v. sensitive)

b) BAL: for TB bacilli (if no sputum is available).

3. Tuberculin test: (Mantoux test)

a) Method:

- Injection of 0.1 cc of 1/10,000 PPD (Purified Protein Derivative):
ID in the upper part of the front of the forearm.
- The result is recorded after 72 hours.
- **Positive tuberculin:** indurated area of 1 cm or more in diameter.

b) Values:

1. Positive test indicates: (adult)

- Infection with TB (whether active or old).

2. Positive test in children below 3 years usually indicates:

- Active infection.

3. Negative test usually indicates:

- Absence of infection with TB.

4. Negative test in adults susceptible to infection (Med, Paramed) indicates:

- The need for BCG vaccination.

BAL
- Pneumocystis
- ILD
- TB

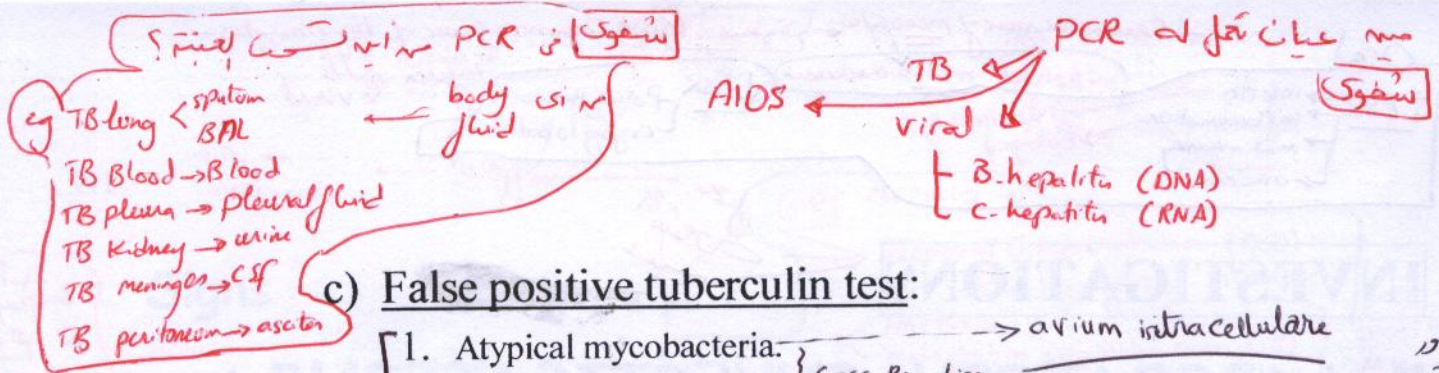
clinical pathol

ID tests

- Tuberculin
- Brucella
- B.A.
- Hydatid (Casoni)
- Sarcoidosis (Kveim)

no damage

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120

- d) False negative tuberculin test: أى حاجه تصحع النتيجة
1. Fulminant cases, e.g. acute miliary TB.
 2. Decreased immunity: Lymphoma, AIDS, Corticosteroids, DM
 3. Inactive tuberculin. المادّة بائنة
- anergy ← تصحع النتيجة
- shadowing TB causes false -ve

4. PCR: (Polymerase Chain Reaction)

- For detection of: Mycobacterial DNA. amplification
 (DNA كثر by replication)

II. IMAGING

1. CXR:

- Radiologic findings are more prominent in the upper lung zones.



- Radiologic findings are variable depending on the lesion:



- Fibrocavitary TB: apical cavities surrounded by opacities.
- TB bronchopneumonia: irregular patches scattered in the lungs. (Fluffy cotton)
- Miliary TB: numerous small shadows of equal sizes (GGA).



4. Others:



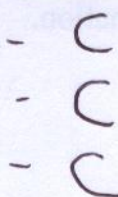
- Fibrosis. heterogeneous opacities & reticulation
- Calcification.
- Enlarged hilar LN. (middle mediastinum)
- Pleural effusion.
- Bronchiectasis [honey combing] free or encysted due to fibrosis
- Carinal LNS
- DD = dumbbell of Gr pulmonary

2. CT scan.

III. BIOPSIES

Bronchoscopy (Transbronchial, Pleura, LN):

- "Typical TB granuloma" (see before).



اشج

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EXTRA - PULMONARY TB

- It occurs due to Hematogenous spread of TB bacilli from:

1. Primary TB: from the pulmonary or glandular component.
2. Postprimary TB: from the pulmonary lesion.

Apical cavity
fibrosis

CLINICAL PICTURE

Single organ affection

1. Thoracic:

- a) Heart: e.g. pericardial effusion, constrictive pericarditis.
- b) Mediastinum: e.g. TB lymphadenitis.
- c) Chest wall: e.g. ribs (osteomyelitis).

2. Extra - Thoracic:

- a) Lymphatic:
 - TB lymphadenitis.
- b) CNS:
 - TB meningitis. ++ICT
 - Tuberculoma.

c) Abdominal:

- TB enteritis:
- TB peritonitis.

single multiple } according to immunity level

Chest Ejaundice
- COPD: RSHF

Causes of jaundice
① Anti TB drugs
② Liver affected by TB

① → Porta Hepatis LNS → obstr. jaundice
② → Paraaortic LNS → TB is 2nd & last common cause of Paraaortic LNS
③ → epitrochlear LNS → not → malignancy

generalized LNS enlargement

Clinical long case

v. difficult D.D

TB Crohn's disease

commonly in the ileocecal region.

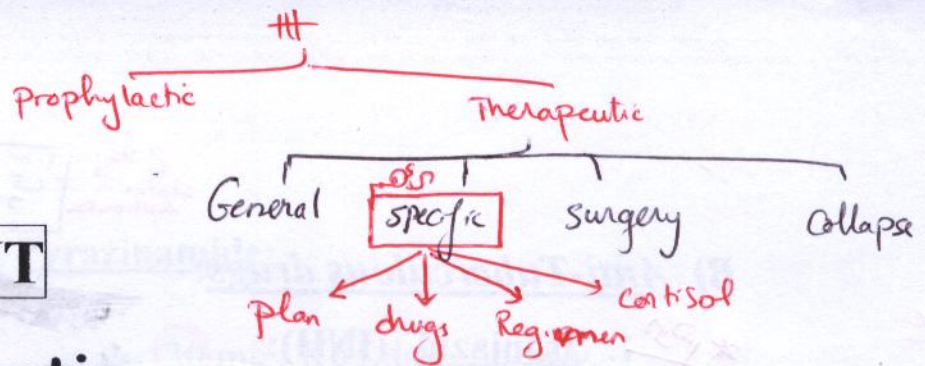
Causes of Epitrochlear LNS enlarg

Syphilis
Sarcoidosis
Leprosy
Lymphoma
TB
Brucellosis

most C. cause of ascites
• liver cell failure
Pleural TB & Pericardial

most C. cause of meningitis
• meningococci

TREATMENT



I. Prophylactic

1. Early diagnosis: (for high risk groups, e.g. Med & Paramed)
 - Mass radiography.
 - Tuberculin survey.
 - Sputum examination.
2. Immunoprophylaxis: (for high risk groups, e.g. Med & Paramed)
 - BCG vaccination. *(active immunization)*
3. Chemoprophylaxis: (for contacts of tuberculous patients)
 - INH. *(passive immunization)*

II. Therapeutic

1. General measures

- Proper nutrition.
- Rest in bed: *in acute cases.*
- Drugs: *antipyretics, expectorants, mucolytics.* *(as it's numular)*

2. Specific measures

"Chemotherapy"

A) Plan of therapy:

لازم کنا واحد

1. Combined drug therapy is needed to:

1. Overcome natural resistance to anti - TB drugs.
2. Accelerate mycobacterial clearance.

لازم طویل

2. Long duration of therapy is needed especially in:

- Extrapulmonary TB.

Months uptill year

B) Anti-Tuberculous drugs:

علاج
Stalin
asthma

سفي

حفظ زي اسل

1st line

5
10
15
25
30

oral mg/Kg/d
IM

* 1. Isoniazid (INH):

Dose:

- 5 mg / Kg / day orally.

Side effects:

- LIVER: Hepatotoxicity (CAH).
- NEUROLOGICAL: (mainly sensory, give B6).
 - PN
 - Psychosis & epilepsy.
- LUPUS - LIKE manifestations.

Chronic active Hepatitis & cirrhosis

oral: to regenerate the liver (النسيج الكبدي)

most common cause is DM

* also
α-methyl dopa

* also Lidocaine: confusion & convulsions

* also
- hydralazine (anti-HTN)
- Procainamide class IA antiarrhythmic

* 2. Rifampicin:

Dose:

- 10 mg / Kg / day orally.

Side effects:

- LIVER: Hepatotoxicity. (reversible) no CAH no cirrhosis
- GIT: irritation.
- URINE: red colouration.

also
digoxin
cortisol

Oral
- anti TB

- antibiotic
- prophylactic not curative in meningitis
- curative in Brucellosis
- anti-leprotic - Rif & Dapsone

نبتة الحية
الحجر. نبتة الحية

3. Streptomycin:

Dose:

- 15 mg / Kg / day

Side effects:

- RENAL: Nephrotoxicity.
- OTOTOXICITY: vertigo, deafness.
- NEUROLOGICAL: ataxia, nystagmus.

IM.

الوحيد في كل
anti TB → IM
if given orally only
it acts locally on GIT

anti TB
&
also
Antibiotic
(aminoglycosides)

4. Ethambutol:

Dose:

- 25 mg / Kg / day orally.

Side effects:

- EYE: optic neuritis.

Toxic on cerebellum
rhythmic oscillation
due to cerebellar ataxia

may cause blindness

5. Pyrazinamide:

Dose:

- 30 mg / Kg / day orally.

Side effects:

- LIVER: Hepatotoxicity. (reversible) as Rif
- METABOLIC: Hyperuricemia.

2nd line
used rarely

1 gm,
not mg

6. Ethionamide:

Dose:

- 1 gm / day orally.

Side effects:

- LIVER: Hepatotoxicity.
- GIT: irritation.

also
Thiazide
& loop
& diuretic (VO)
↑ uricemia
↑ glycaemia

INH
Rif
Pyrazinamide
Ethionamide

Cyclosporin (action)
biphenyl
Cyclophosphamide

7. Cycloserine:

Dose:

- 1 gm / day orally.

Side effects:

- NEUROLOGICAL: dizziness, depression.

8. Para-aminosalicylic acid (PAS):

Dose:

- 10 gm / day orally.

Side effects:

- GIT: irritation.
- HYPERSENSITIVITY: e.g. skin rash.

INH
strepto
Cycloserine

aminoglycosides

9. Capreomycin, Kanamycin, Amikacin:

Dose & side effects: as streptomycin.

علامات القصور

- CLP → نسق
- Sputum → -ve
- CXR → نسق

C) Regimens:

1st 2M : 3 drugs
Rest : 2 drugs

1. Short regimen: (9 – 12 months)

- INH & Rifampicin: for 9 – 12 months.
- Ethambutol plus Streptomycin: for the first 2 months only.

2. Very short regimen: (6 months)

1st 2M : 4 drugs
Rest : 2 drugs

- INH & Rifampicin: for 6 months.
- Ethambutol plus Streptomycin: for the first 2 months only.
- Pyrazinamide: for the first 2 months only.

3. Classic regimen: (Rarely used now: 15, 18 or 24 months)

- INH & Rifampicin: for the whole duration.
- Ethambutol plus Streptomycin: for the first 2 months only.

D) Indications of corticosteroids in TB:

سفوكي شهور - Corticosteroids are given under cover of anti-TB drugs in:

1. TB meningitis.

2. TB serositis

(pericardial, pleural, peritoneal).

3. TB adrenal glands

(replacement therapy).

4. Fulminant cases,

e.g. miliary TB.

3. Surgery:

(resection)

- Tuberculoma. (in brain for eg)
- Destroyed tuberculous lobe.



4. Collapse therapy. obsolete.

Most common carcinoma
 Br. Ca. → aggressive
 Hepatocellular Ca. →
 Br. Ca. →
 Breast →
 Br. Ca. →
 Case 11

Br. Ca. → aggressive
 Hepatocellular Ca. →
 Br. Ca. →
 Breast →
 Br. Ca. →
 Case 11

Smoking on body
 لا يمكن القلي
 • Cancer: Lip, Tongue
 Pharynx, larynx
 127 Bronchi
 • Cancer: GIT, U.B.
 • Chronic Bronchitis
 COPD

BRONCHOGENIC CARCINOMA

INCIDENCE

- The most common malignant tumour in males in most of the world (due to smoking).
- More common in males (due to smoking).
- More common between 50 & 70 years of age.

PREDISPOSING FACTORS

1) Cigarette smoking:

- 90 % of cases of bronchogenic carcinoma are caused by smoking cigarettes.
- Quantity x duration = Pack x years (both are risk factors).

2) Occupational exposure: (miners)

- Asbestos, nickel, uranium.

3) Chronic lung inflammation, e.g.

- Interstitial fibrosis (ILD)

30y x 20 pack 600
 20y x 30 pack 600

Pneumococcosis
 TB
 Pre cancerous

Asbestos → Adenocarcinoma

Chronic irritant → Carcinoma

Adeno Ca.
 Asbestos non
 Smoker

PATHOLOGY

1 Types:

- Bronchogenic carcinoma is divided according to the characteristics of the disease and its response to treatment into:

1) Small cell carcinoma

- Highly malignant & rapidly growing.
- The most common to produce paraneoplastic syndrome.
- The only one to respond to chemotherapy.
- v.v. bad prognosis

نبي الشوفان

(oat cell carcinoma)

20%:

000

مفرجة
 نبي نبات الشوفان
 الزنج جدي لوفنت تحت ميكروبي

2) Non Small cell carcinoma

(classified histologically into):

- Squamous cell carcinoma: 40 %
- Large cell carcinoma: 15 %
- Adenocarcinoma: 25 %
 - Mostly common in asbestos exposure.
 - Mostly common in non-smokers.
 - Mostly common in females.

d) Alveolar cell (bronchiolar) carcinoma: 1 %:

- Low grade carcinoma that presents as an alveolar infiltrate.

from terminal Bronchiole
 or " alveoli

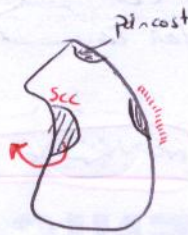


كله من هنا

ال

alveolar
 هنا
 أو
 هنا

وده
 نبي



2 Site:

- Bronchogenic carcinoma is divided according to its site of origin into:

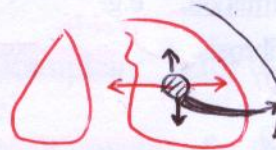
	Central (hilar)	Peripheral
Incidence	More common	Less common
Type	Squamous cell carcinoma	- Adenocarcinoma - Large cell carcinoma
Spread	To mediastinum early	To pleura early
Clinical picture	- cough, hemoptysis - dyspnea, chest pain	pleurisy
Bronchoscope	Easily visible	Not visible

- Pancoast tumour is an apical peripheral carcinoma. esp. as Pancoast tumor

3 SPREAD

1) Direct:

- To: other parts of the lung.
- To: pleura & mediastinum.
- To: ribs & vertebrae.



2) Lymphatic:

- To: hilar & mediastinal lymph nodes.
- To: cervical, axillary, abdominal lymph nodes.

middle of the 4

Porta Hepatis → obstr. jaundice
Para aortic, coeliac
mesenteric

NB Retrograde spread along the lung lymphatics may occur leading to lymphangitis carcinomatosa which may lead to subacute cor pulmonale.

3) Blood:

- To: liver.
- To: brain.
- To: bones.



GGA

- 1 - recurrent shadows
- 2 - lymphatic carcinomatosis

lung
no presentation

Type of pt: ♂, old, heavy smoker

CLINICAL PRESENTATIONS & MANIFESTATIONS

1) ASYMPTOMATIC PRESENTATION



- There are no symptoms, but a coin shadow is discovered accidentally on CXR. *early small round Routine checkup*
- & 1st presentation can be metastasis *اكتشفه من*

no presentation
لا توجد أعراض
أول عرض
قد يكون انتشار

Causes of fever in Br. Ca

- ① Bronchiectasis (Br. Ca → obstruct → retain secretions)
- ② Abscess
- ③ ~~Pneumonia~~ Pneumonia
- ④ empyema (rupture abscess)
- ⑤ Unexplained (Paraneoplastic Sydn)

تپری لپا
و سوزش

2) CHEST PRESENTATIONS

A) Broncho-pulmonary presentations:

1. Cough & hemoptysis.
 - ① Hge & necrosis
 - ② Neovascularization (V. fragile)
2. Bronchial obstruction:

a- Partial: causing:

- Localised wheeze persistent after cough.
- Localised bronchiectasis.

b- Complete: causing:

- Collapse which may be complicated by lung abscess.

3. Pneumonia:

unresolving, or recurrent.

4. Lung abscess:

due to:

1. Secondary infection in a collapsed area.
2. Breakdown of the tumour.

5. Diaphragmatic paralysis.

Inspection
Percussion
CXR Screen

-ve (absent) Litten sign
Reversed tidal percussion
Paradoxical movement

Apex
- Pancoast
- TB
- Klebsiella

B) Thoracic inlet or superior sulcus syndrome:

- Occurs in cases of pancoast tumour, & manifestations are due to involvement of:

1. The lower trunk of the brachial plexus, (C8, T1) causing:

- o Pain along the medial aspect of the arm, forearm & hand.
- o Weakness & wasting of the small muscles of the hand.

2. The sympathetic chain, causing:

- Horner's syndrome. unilateral ptosis

3. The blood vessels at the root of the neck, causing:

- A. • Unequal pulse & BP (arterial obstruction) & ischemia
- V. • Congested non-pulsating neck veins & dilated veins over the chest & cyanosis (venous obstruction).

face u.l. neck of innominate v.v.

normally

serial withdrawal
of I.C. space
(suction)
in thin pt, good
illumination
due to diaphragm
descent
absent in

D. Paralysis
P. effusion

Both are in
Br. Ca.

eff lid

open by III
close by V
Other cause
unilateral
ptosis
in III
paralysis

C) Pleural presentations:

1. Dry pleurisy:

- Due to infiltration & irritation of the pleura.

2. Pleural effusion:

of different types may occur:

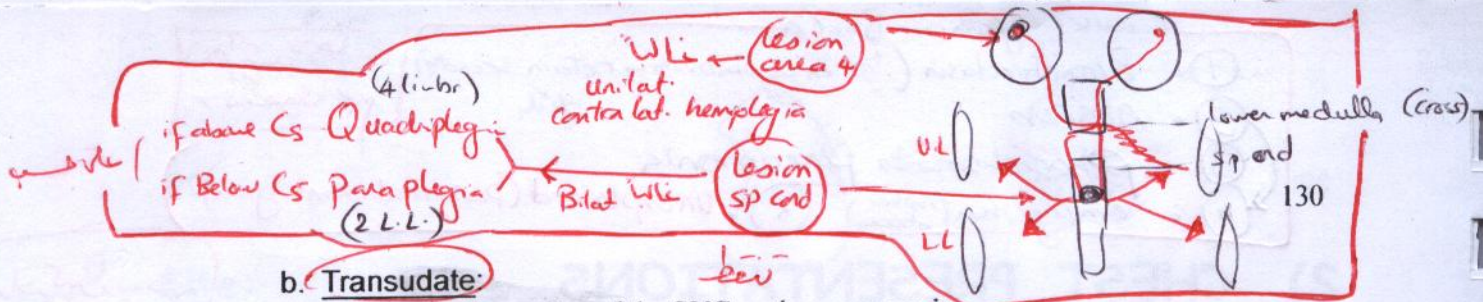
-ve Litten sign

a. Exudate (Malignant effusion): the most common type

- Hemorrhagic, massive, rapidly re-accumulating after aspiration. & malignant cell
- The mediastinum may be shifted to same side of effusion due to underlying lung collapse.

⑤

للال
تتراسیکلین



b. Transudate:

- Due to obstruction of the SVC or the azygos vein.

c. Chylous effusion:

- Due to obstruction of the thoracic duct.

d. Suppurative effusion (Empyema):

- Due to rupture of lung abscess into the pleura.

• esoph.: dysphagia

• RLN: Hoarseness voice

D) Mediastinal presentations:

- The patient may present with features of “Mediastinal syndrome” due to:

- o Direct effect of the tumour.
- o Mediastinal lymph node metastasis.

راجل کیرج اسن پیدخه
مقاله صوتی تغییر
اقله

3) METASTATIC PRESENTATIONS

a. Direct metastasis:

- Erosion of the ribs & vertebrae.

b. Lymphatic metastasis:

- Lymph node enlargement.

c. Blood metastasis:

- Liver: enlarged, hard, tender, irregular liver.
- BMS: ↑ ICT & focal neurological manifestations.
- Bones: bony pains & pathological fractures.

Blurring vision
headache
Projectile vomiting

eg: area 4 (motor) in
Frontal lobe
Contra lat. hemiplegia

or sp. cord →
Paraplegia (if below)
Quadr. plegia (if above)

osteolytic
(Osteoclast)

canon
prostate
osteoblastic
metastasis

4) NON-METASTATIC PRESENTATIONS

“Para-malignant syndrome”

- These manifestations are most commonly associated with small cell carcinoma.
- The underlying mechanisms of these manifestations are unknown.
- The endocrinal manifestations ONLY may be explained by the abnormal secretion of ectopic hormones from the tumour.

These manifestations include:

1. Neurological:

- o Encephalopathy (subacute cerebellar degeneration).
- o Peripheral neuropathy, myopathy, myasthenia gravis.

2. Endocrinal:

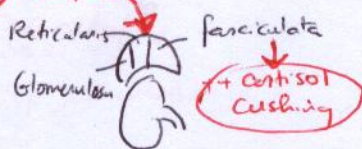
- o Hyperparathyroidism.
- o Hypoglycemia.
- o Cushing's syndrome.
- o SIADH
- o Gynecomastia.

not ++ cortisol but ++ ectopic ACTH
(retention of water & hyponatremia).

Syndrome of Inappropriate ADH (post pit)

130

renal tubules
water reabsorption
کثیر



حوار سق

Q: Pneumo
A: Unilat. ptosis

Q: why
A: infiltrative synp (Horner)

Q: Bilat. ptosis

A: yes in Myasthenia gravis

Q: why

A: unknown

ماثلف

مفشی غیر

یوکل اکلوت

Oat

PN
metastasis

Bilat. ptosis
NMJ

Water/KnightLove

Acanthosis nigricans

- 1- Color: dark brown or black
- 2- Site: skin of neck or axilla or anogenital region
- 3- Consistency: velvety
- 4- Caused by $\left[\begin{array}{l} \text{Br. Ca} \\ \text{Stomach Ca} \end{array} \right]$ غرورون
Tyrosinemia : hyperinsulinemia (due insulin Resistance) 131

3. Hematological:

- o Anemia.
- o DIC.

procoagulants or prothrombotic (due to release of $\text{TNF}\alpha$)

4. Dermatological:

- o Dermatomyositis.
- o Herpes zoster.
- o Acanthosis nigricans.

Abs from tumor (مخبر) $\rightarrow$ against Hs & skin

lives normally in $\text{I} \& \text{Inter}$ if $\downarrow$ imm $\rightarrow$ activates

5. Skeletal:

- o Clubbing.
- o Hypertrophic osteoarthropathy.

chronic toxemia $\left\langle \begin{array}{l} \text{Abscess} \\ \text{Bronchiectasis} \end{array} \right.$

non toxic non hypoxic
Painful. قسطنطين
هو غير مفسر

6. Metabolic:

- o Cachexia. (unknown) maybe due to cachexin secretion
- o Fever. (unexplained) $\leftarrow \text{TNF}\alpha \rightarrow$ (catabolism)

also

RSHF

Cardiac
cachexia

INVESTIGATIONS

1) Chest X Ray: (v v v very important)

- It is the most valuable easy investigation for bronchogenic carcinoma.
- It detects only tumours bigger than 2 cm in size.
- It may show:

• Lung:

- o Coin shadow or a big mass.
- o Pneumonia, lung abscess, or collapse.
- o Pancoast tumour: apical mass.

• Mediastinum:

- o Mediastinal mass: it could be the tumour itself or its lymph nodes.

• Pleura:

- o Pleural effusion, with mediastinal shift to the same side. due to collapse

• Paralyzed diaphragm:

- o Elevated cupola on the affected side.
- o Paradoxical movement on the screen.

• Chest wall:

- o Erosion of the ribs or vertebrae.

Pathological fracture

no fracture + pl. eff

or

tumor

trauma
 $\downarrow$
pl. eff.

$\downarrow$
pl. eff.

لا تسمى infective ولا Bacteriology
لا تسمى virus ولا Biotry ← ده كلام، لست
وهذا كلام غير على
Disseminated for biopsy

Investigation

2) CT chest:

- It is very accurate & sensitive in detecting the tumour.
- It can detect small tumours (smaller than 2 cm in size) that do not appear in the ordinary Chest x ray.

BAL ۱۰۰

Pneumocystis
ILD
TB
Br. Ca.

3) Bronchoscopy & Biopsy:

- Visualization & localization of the tumour, especially the central type.
- Biopsy could be taken for pathological examination.
- BAL could be obtained & examined for malignant cells.

4) Cytological examination:

for detection of *malignant cells*

1. Sputum examination:
2. Aspiration of pleural effusion:
3. Biopsy:

may be positive for malignant cells.

may be positive for malignant cells.

- o *Transbronchial:* through bronchoscopy.
- o *Lung biopsy:* open or percutaneous
- o *Lymph node biopsy:* from enlarged lymph node.

3. Biopsy

guided by C.T.
resolute $\uparrow$ $\sim$ the

DIFFERENTIAL DIAGNOSIS

A) Clinically:

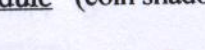
- The possibility of bronchogenic carcinoma: should be considered in any old, chronic heavy smoker male with a chest complaint. until P
- The DD of bronchogenic carcinoma includes:
 1. Other causes of prolonged cough or hemoptysis. eg
 2. Other causes of pleural effusion. eg : TB
 3. Other causes of localized bronchial obstruction. eg: out : wall lumen : FB.
 4. Other causes of mediastinal syndrome. eg:
 5. Tuberculosis.
 6. Unresolving pneumonia or lung abscess. eg : Bronchocetosis

Until Proved otherwise

Causes any chest lesion

TB
8 Br. Co.
should
be considered
D.D.
in any chest
lesion

B) Radiologically:

- Differentiation from other causes of solitary pulmonary nodule (coin shadow):
 1. Pulmonary tumours:
 - Benign tumour. *Bronchial adenoma*
 - Malignant tumour.
 - Solitary metastasis.
 2. Pulmonary infections, e.g. TB, pneumonic patch.
 3. Pulmonary infarction.
 4. Foreign body.
- 

4. Foreign body.
5- phantom & tumor : encysted pl. eff.
disappears by diuretics

also in
neuro
MS (DS)
multiple sclerosis
Disseminated
↓
O.D.
gany neuro

يطلب

TREATMENT

- *Successful surgery offers the only chance of complete cure of bronchial carcinoma.*

- *Choice of treatment depends on:*

1. Histological type of the tumour.
2. Operability:
 - o No evidence of hilar or mediastinal involvement.
 - o No evidence of extra-thoracic spread.

- *Lines of treatment include:*

1. For non-small cell carcinoma:

A) Operable:

- Pneumonectomy or lobectomy which may be followed by postoperative irradiation.

B) Non-operable:

- Chemotherapy & palliative radiotherapy.

2. For small cell carcinoma:

- Chemotherapy: e.g. cyclophosphamide.
- Radiotherapy: for treatment of the primary tumour & prophylaxis against cerebral metastases.

Neurological manifestations of bronchogenic carcinoma

نظريه
حاصل دلك

1) Chest presentations:

Broncho-pulmonary presentations:

- Diaphragmatic paralysis.

Thoracic inlet or superior sulcus syndrome:

- Occurs in cases of pancoast tumour, & manifestations are due to involvement of:

The lower trunk of the brachial plexus, (C8 T1) causing:

- Pain along the medial aspect of the arm, forearm & hand.
- Weakness & wasting of the small muscles of the hand.

The sympathetic chain,

- Horner's syndrome.

causing:

unilat
ptosis

Mediastinal presentations:

- RLN paralysis, causing hoarseness of voice.

2) Metastatic presentations:

- ^{CNS} Brain: ↑ ICT & focal neurological manifestations.

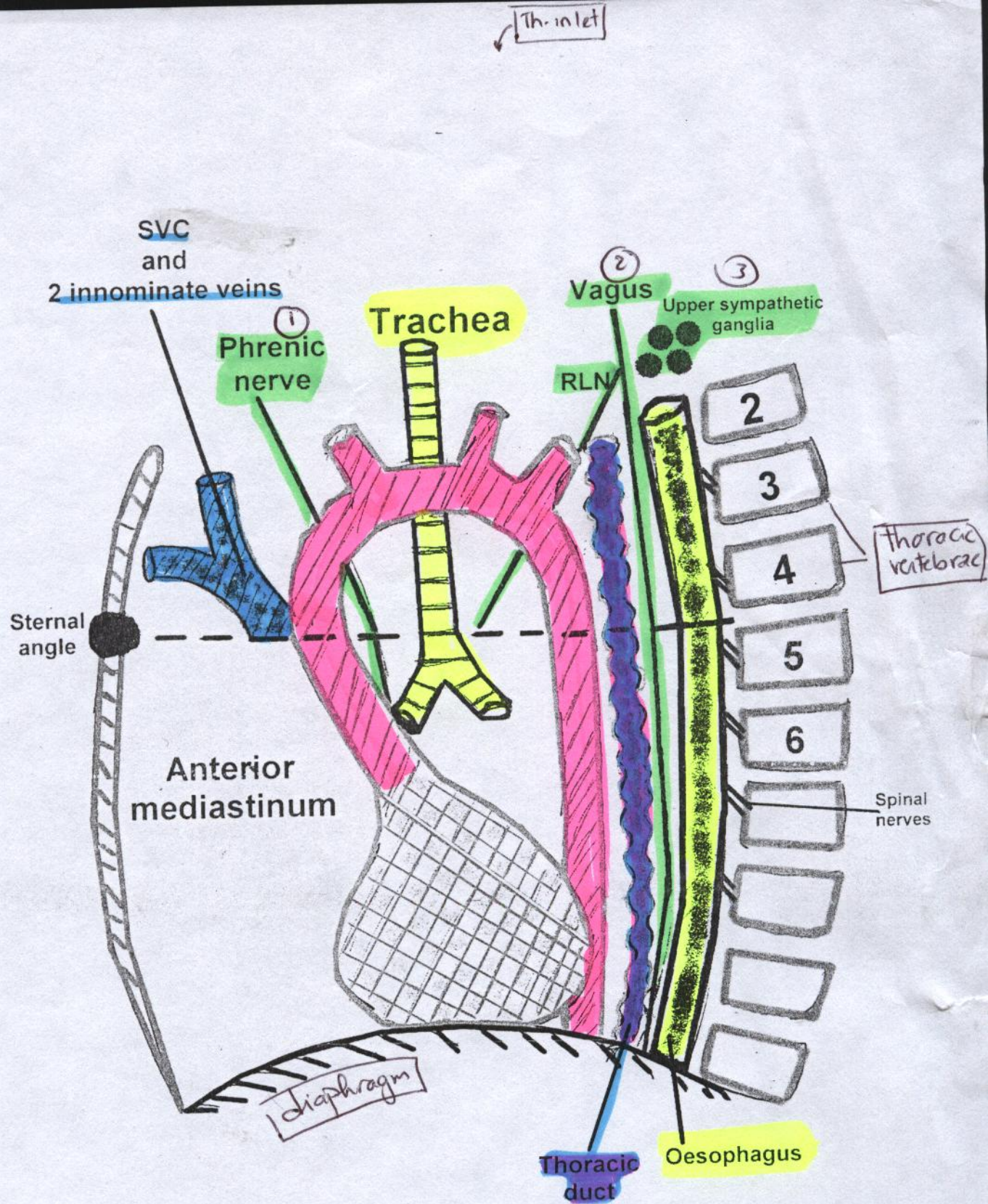
3) Non-Metastatic presentations:

"Para-malignant syndrome"

- Encephalopathy (subacute cerebellar degeneration).
- Peripheral neuropathy, myopathy, myasthenia gravis.

- Herpes Zoster

Ant.



نظري + 100%

ANATOMY OF THE MEDIASTINUM

1- The mediastinum is the ^{فراغ} central part of the thorax. *betw 2 lungs*

2- The mediastinum has the following boundaries:

- Anteriorly: *the sternum.*
- Posteriorly: *the thoracic vertebrae.*
- Superiorly: *the thoracic inlet.*
- Inferiorly: *the diaphragm.*
- Laterally: *the parietal pleura.* *(2 lungs m)*

3- The mediastinum is divided by an imaginary line into:

1 • Superior mediastinum.

2 • Inferior mediastinum.

The line passes from the sternal angle ^{*} to the lower border of the 4th thoracic vertebra ^{*}

SPine of T2

*below w
if Bronchial
Breathy
= Deep
sig*

- The inferior mediastinum is subdivided by the pericardial sac into:

- Anterior mediastinum.
- Middle mediastinum.
- Posterior mediastinum.

CONTENTS OF THE MEDIASTINUM

I. Superior mediastinum:

1. Vessels:

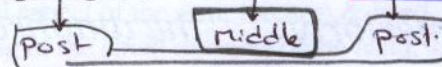
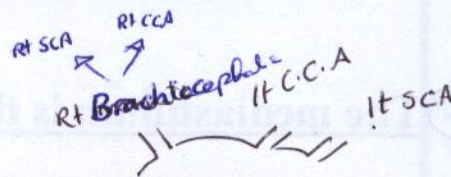
- Arteries: Arch of the aorta & its 3 big branches. Post
- Veins: ★ SVC & the 2 innominate veins.

2. Tubes:

- Upper parts of: oesophagus, trachea, thoracic duct.

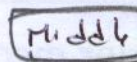
3. Nerves:

- Vagus & Left RLN.
- ★ Upper sympathetic ganglia.
- Phrenic nerves.



بقاوا في

Autonomic nerves



inferior

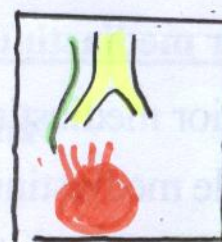
II. Anterior mediastinum:

- Thymus gland.
- Fat.

LNS in all
mediastinum
esp
superior
& middle

III. Middle mediastinum:

- Pericardial sac & its contents (heart & its big vessels).
- Trachea & the main bronchi.
- Phrenic nerves.



Ascending Ao.
pulm. A.

LNS

IV. Posterior mediastinum:

1. Vessels:

- ١٤٧ (+) Arteries: Descending aorta.
 ١٤٧ (+) Veins: Azygos & hemiazygos veins.

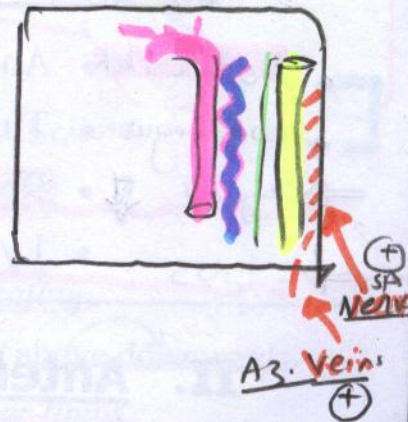
2. Tubes:

- ١٤٧ (+) Oesophagus.
 ١٤٧ (+) Thoracic duct.

3. Nerves:

- ١٤٧ (+) Vagus nerve.
 ١٤٧ (+) Spinal nerves.

~ m. v. p.
vertebrae



NB

Lymph nodes are present in different parts of the mediastinum.

esp sup
& middle → carenal
= (Hilar)
= (mediastinal)

anatomy of mediast
manif of Mediast. Syndr

سؤال مؤكد نظري
وهو ده سؤال anatomy لوحده
في هذا الكتاب

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MEDIASTINAL SYNDROME

1 ETIOLOGY

- غالباً عبارة عن مكونات وسائط سريرة (Goitre)
- غالباً الريبوسايدوم على المكونات وسائط رتق على حافة سريرة
as Brachial plexus

I. In the Superior mediastinum:

- Aortic Arch** • Aneurysm of the arch of the aorta or one of its 3 big branches.
- esophagus** • Tumours.
- Thyroid** • Retrosternal goitre.
- LNS** • Lymphadenopathy. — lymphoma, leukemia, Br. Ca, TB, Sarcoidosis

II. Anterior mediastinum:

- Thymoma** • Oral • طلع من فيه
Oral : pressure manifestation
it's fn is T-cells matured if tumor → Myasthenia gravis • تفاعل في حافة سريرة

III. Middle mediastinum:

- Asc. Ao, Pulm. A.** • Aneurysm of the ascending aorta or the pulmonary artery.
- Heart** • Pericardial effusion. or any cardiomegaly
- Bronchi** • Bronchial carcinoma.
- LNS** • Lymphadenopathy.

IV. Posterior mediastinum:

- Desc. Aorta** • Aneurysm of the descending thoracic aorta.
- esophagus** • Oesophageal tumours.
- Stomach** • Hiatus hernia. • Stomach n°
- Vagus spinal** • Neurofibroma.
- N. N.**

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②

CLINICAL PICTURE

I. Manifestations of the cause

eg: Pericardial effusion
eg: Br. Carcinoma

II. Pressure manifestations:

1. Pressure on vessels:

A) Branches of aortic arch:

- Unequal pulse & BP in the upper limbs. (subclavian A.)
+ arterial ischaemia

B) SVC:

1. Neck veins: congested non-pulsating.
2. Dilated veins: on the chest wall filling from above downwards.
3. Oedema & cyanosis: face, neck & upper limbs.
drained by SVC



2. Pressure on tubes:

A) Oesophagus:

- Dysphagia.

by < Pericardial eff
LAE

B) Trachea & bronchi:

- Dyspnea, stridor, if pressure on upper trachea
- Cough (brassy). Low, harsh, due to irritatn
- Wheeze (localised).

C) Thoracic duct:

- Pericardial effusion.
- Pleural effusion.
- Peritoneal effusion (ascites).

Chylous

Rupture obstruction

Br. ca. infiltrates syp. ganglion → unilat ptosis

Unilat 2 causes → Cr. III (Emydrasia)
Hornor (Ert. osin)

Bilat 2 causes → Myasthenia
Congenital

Bilat. Ptosis

Mediast. Syndrome

Br. ca. Myasthenia gravis

rep gain

140

3. Pressure on nerves:

A) Vagus or left RLN:

- Hoarseness of voice.

B) Sympathetic chain:

- Horner's syndrome.

Autonomic nerves

ptosis
Miosis
enophthalmos
anhydrosis

C) Phrenic nerve:

- Diaphragmatic paralysis.

D) Brachial plexus:

(lower trunk)

- Pain along the medial aspect of:
- Weakness & wasting of:

apex of the lung
by PanCost tumor

arm, forearm, hand (C8, T1)
small muscles of the hand.

(C8, T1)

E) Intercostal nerves:

- Intercostal neuralgia.

as LMNL
root
AHC
not LMNL

LMNL not cause wasting
except v.v. late

4. Pressure on bones:

(Ribs, sternum, spine)

- Bone pains.

Infiltration (v.v. late & terminal)

3 INVESTIGATIONS

I. Imaging:

1. CXR:

- To detect the mediastinal mass (size & site).

2. CT & MRI chest:

- More accurate in diagnosing the mass.

3. Fluoroscopy (=Screen)

- To detect the position & mobility of the diaphragm.
- To detect pulsations of an aneurysm.

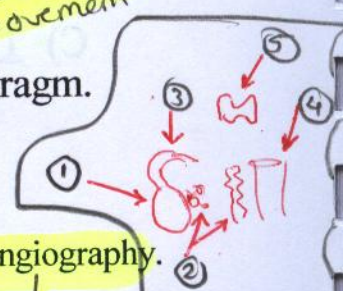
4. Others:

- Echocardiography, Ba swallow, thyroid scan, lymphangiography.

passively upwards
actively downwards



Paradoxical movement



140

eg. ktb kl
2, 3, 4

eg P.E

Chamber enlargement

eg esoph.

angiograph
D uplex

eg Th. duct
LNS

eg aneurysm

White Knight Love

II. Endoscopy:

- Bronchoscopy.
- Oesophagoscopy.
- Mediastinoscopy. *دس، س، ف، م*

III. Exploratory thoracotomy.

الفتح

④

TREATMENT

- TTT of the cause.

*eg surgical removal of RSG
(Retrost. gall)*